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THEORIES on ALCOHOLISM

Edited by

C D Chaudron and D A Wilkinson



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**THEORIES on
ALCOHOLISM**

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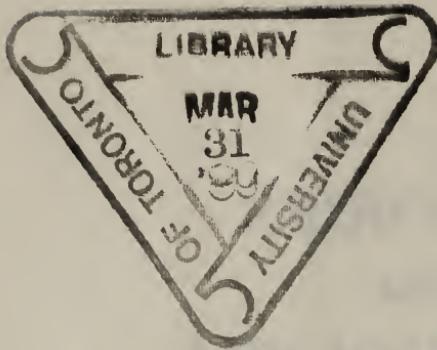
edited by

C. Douglas Chaudron and D. Adrian Wilkinson

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Toronto, Canada**



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FOREWORD

Don Cahalan

The goal of this book is to serve as a methodological and epistemological resource for serious students of the dynamics of addiction, as well as for practitioners who treat alcoholics and the policymakers who have to cope with a myriad of controversial legal, public health, and medical care issues. During the last several decades there have been many books published on a range of theories related to alcoholism; however, this is the first in which the editors have taken considerable pains to recruit authors from a wide range of disciplines with different theoretical perspectives and to require that they follow specific guidelines on organization and style in their chapters, so that the discussions of the different theoretical positions can be compared by the reader within a consistent framework.

This book is divided into eleven chapters organized into three sections: biological theories (including genetic, neurobiological, and neurobehavioral aspects), psychological theories (psychoanalytic, personality, classical conditioning, and social learning), and social theories concerning alcoholism (systems, availability, anthropological, and economic aspects). The editors did not impose a single, narrow definition of "alcoholism" upon the authors of the individual chapters, but let them define the dependent variable as they pleased. As the editors say in the Introduction, the disease concept of alcoholism and the alcohol dependence syndrome are not here analysed as theories *on* alcoholism, these being viewed as descriptive rather than explanatory theories of alcoholism not fitting within the formal objective of this book, which is to emphasize that alcoholism is a multiply determined biopsychosocial phenomenon. Also, the editors note that several other theories on alcoholism were not addressed because of a dearth of sufficient research to evaluate their worth. These include humanistic psychology, existential theories, and Marxist perspectives.

The contributors are all specialists, with many years of experience in alcohol-related studies, drawn from a very wide range of disciplines. All authors were encouraged to be as objective as possible in dealing with the explanatory power of the theories they were to discuss. Since most authors are dealing with theories associated with their own discipline, readers may expect that the theories that relate to the writer's professional interests might be treated with more respect and understanding than theories from other fields; thus readers should suspend judgment on the relative explanatory power of the various theories on alcoholism until they have read the whole book and have had full opportunity to make a critical analysis of the various theories. In any case, as the editors emphasize, in dealing with such a multifaceted phenomenon as alcoholism, the central objective of the book is to encourage

readers to visualize how the various theories can be *complementary* to each other in explaining alcohol-related behavior, rather than competing; it is hoped that the field of alcohol studies has long since graduated from the single-cause polemical battles of the past.

Now to summarize some of the implications of the individual chapters:

THE ELEVEN THEORIES ON ALCOHOLISM

Chapter 1, on genetic theory: Unlike in most of the other chapters, the author here is highly critical of the explanatory power of the theory under discussion, primarily on the grounds that the past research (particularly the studies of twins) has contained errors and lacked appropriate controls and documentation. Lester's critique of the much-in-vogue genetic theory is detailed and thoroughgoing and should stimulate further re-examination of the evidence. A number of his points about the twin studies are discussed independently in a parallel critique by Fillmore (in press), who points out that emphasis on genetic factors in alcoholism unduly plays down the myriad of other sociopsychobiological forces affecting the development of problems. It remains to be seen whether Lester's questions about the worth of the genetic theory will serve as a brake on the current enthusiasm for it among some alcohol program administrators, not to mention representatives of the liquor industry, who find the theory useful because it directs attention back upon the alleged genetic deficiencies of the (presumably relatively few) afflicted alcoholics and away from the role of alcohol as the agent in the development of alcoholism and other alcohol-related problems.

Chapter 2, on neurobiological theory, is a detailed presentation of the evidence regarding the role of neurobiological reinforcement, tolerance, and physical dependence in the development of alcoholism. It discusses some implications of the theory for treatment, which should be helpful in deciding whether the individual should be counselled that total abstention appears to be the only workable goal, or that tolerance has not advanced to the point that would preclude consideration of controlled drinking as at least an interim goal.

Chapter 3, on neurobehavioral theory, abundantly documents the association between neuropsychological anomalies and later alcoholism. However, as in Chapter 1, the authors emphasize the need for caution in conclusions about genetic effects. They also point out that "For alcoholism to develop, alcoholic beverages must be reasonably accessible in society. Facilitative influences, such as ambivalence about drinking by parents and peers, as well as by the socioeconomic and cultural macrosystems, also affect drinking behavior, and ultimately the risk for becoming an alcoholic" (p.82). The authors also emphasize the need for more true longitudinal studies to measure the influence of neurobehavioral status upon later development of alcoholism.

The author of Chapter 4 sees "serious limitations" in the psychoanalytic theory of alcoholism because of its reliance on inference rather than direct observation, as well as the complex nature of psychoanalytic concepts and the differences in their use by various analysts. However, he does commend it for helping to explain some short-term pleasure-seeking behaviors that

ordinarily would be considered irrational because of their self-destructiveness, and he notes how psychoanalytic theory has helped in improving social policy and child-rearing practices. One might add that the psychoanalytic emphasis upon the role of denial and purposive forgetting in alcoholics' resistance to changes in their lifestyles often has been useful in treatment.

Chapter 5, on personality theory, is written in a style suitable for the lay reader as well as the specialist: for example, the author defines "personality" as "the organized pattern of a person's behavioral and emotional characteristics that distinguishes him or her from other people" (p.143). He provides a useful review of the many fluctuations in the emphasis upon personality factors in alcoholism that have occurred since the time of Freud (who had little to say about it) and points out that only recently has there been much emphasis upon the existence of various types of alcoholic personalities. He expresses skepticism about the worth of some of the studies on personality that have proliferated since about 1970, largely on the grounds that they have relied too much on the retrospective reports of alcoholics about their prealcoholic characteristics. He also emphasizes the importance of the interaction of other factors with personality, including biological, environmental, and sociocultural influences.

His style of presentation should make this chapter especially interesting to practitioners. Although he does not provide an encyclopedic list of references regarding the role of personality in alcoholism, he recommends several readily available recent reviews.

Chapter 6, on classical conditioning, is a technical and thorough evaluation of the implications of the theory with respect to preferences and aversions for alcohol beverages, alcohol tolerance and craving, and withdrawal. The authors caution that social factors or expectations of drug effects are better models than classical conditioning in explaining the genesis of drinking, and that the theory does not account for constitutional factors or individual differences in conditionability. However, they make a strong case for the importance to behavior therapists of retaining a Pavlovian perspective in understanding alcohol preferences and aversions and cravings, as well as the process of modification or extinction of conditioned behavior.

Chapter 7, on social learning theory, draws heavily on both classical and operant conditioning principles and also emphasizes the fundamental importance of cognitive, symbolic, vicarious, and self-regulatory processes in acquiring or getting rid of addictions. Among the illustrations of how these mechanisms operate is a discussion of how the presentation of drinking in the media may be operating to encourage heavy or risky consumption. The chapter presents a much-documented discussion of the ongoing controversy over controlled drinking as opposed to abstinence as the treatment goal, with the clear implication that controlled drinking should be considered as at least an interim goal for some alcoholics.

In Chapter 8, on systems theory and alcoholism, "Behavior is...conceptualized as being less a reflection of unique personality and psychological variables, and more a response to the vicissitudes of interpersonal, interactional systems.... A systems perspective emphasizes 'wholeness' and the interdependence of elements.... Within a systems framework, causality is

viewed as being circular rather than linear" (pp.290-291). The author's primary emphasis is upon application of a systems approach in family therapy: he presents much evidence on the necessity of including the alcoholics' significant others in modifying the environment to reduce injurious drinking. Readers interested in interactive systems perspectives in relation to alcoholism should also see the writings of those who emphasize the importance of going beyond family therapy to include the larger spheres of peers, the workplace, or the society as a whole, such as Holder and Wallack (1986) in relation to prevention of alcohol problems.

Chapter 9, on availability theory, deals with the proposition that the greater the accessibility of alcohol in a society, the greater the prevalence and severity of alcohol-related problems. The chapter discusses the distribution-of-consumption hypothesis (commonly associated with Ledermann); in which it is contended "that the mean level of alcohol consumption in a society is closely related to the numbers of persons consuming at levels deemed to be of high risk" (p.332). The author presents considerable evidence on the empirical relationship to be found between changes in control measures (such as hours of service, minimum legal age, and alcohol taxes) and subsequent changes in the level of alcohol consumption and of alcohol-related problems.

The general trend in the industrialized world after World War II has been toward liberalization of alcohol controls, with increased consumption and concomitant problems. The author contends that the predominance of the disease concept and the vast expansion of alcoholism treatment during recent decades has gone along with the trend toward slackened controls over availability, but that increased skepticism about the efficacy of present alcohol programs is now forcing policymakers to give a greater emphasis to preventive controls over consumption.

While the author sees availability theory as having at best tenuous connections to other theories, since it is more empirical than theoretical, some might contend that availability theory should be a key component in any comprehensive systems theory (see notes on Chapter 8 above), since availability can be presumed to be a key factor in establishing and maintaining the climate of drinking.

Chapter 10, on emerging anthropological theory and models of alcohol use and alcoholism, is a condensation of the considerable research and many reviews published by the author over more than thirty years. Heath concedes that there is no unitary anthropological theory on alcoholism, although he sees anthropological and sociological studies as having "had an impact out of all proportion to the fewness of investigators involved" (p.403). He interprets social anthropology as subsuming much of sociology and the other social sciences. The chapter presents a thorough discussion of many sociocultural phenomena involved in alcoholism, including deviance labelling, reference-group effects, anomie, Time Out, and ambivalence, and of the single-distribution model (see Chapter 9), the anxiety model, the social organization model, the conflict-over-dependency model, the power model, the symbolic interactionist model, and the socialization and social learning models. The lengthy chapter closes with conclusions on how much we still have to discover about how social learning

affects alcohol consumption and problems, if we are to combat public ignorance on how alcohol problems build up through social reinforcements. He discusses the necessity for better collaboration between psychologists and other social scientists and physiologists; and he emphasizes the need for more well-controlled studies of the natural history of drinking behavior.

Chapter 11, on economic theory, discusses the implications of the distribution-of-consumption model (cf. Chapter 9) and attendant issues of elasticity of consumption in relation to price changes. The authors add that there is need to study not only economic measures that might influence chronic alcoholics, but also the economic implications of the more prevalent and costly effects of excessive drinking as manifest in reduced productivity, drunken driving, and public disturbances. They also call attention to the need to explore alcohol demand relationships for different age and socioeconomic groups—for example, whether the young are more responsive to alcohol advertising than are other groups.

The authors conclude by noting how economists can contribute to theories on alcoholism and alcohol problems by providing formal models for dealing with alcohol use and misuse, and by providing statistical tools (such as econometric methods and cost/benefit analyses) to evaluate the effectiveness of control programs and policies.

IMPLICATIONS FOR THEORIZING ON ALCOHOLISM

Explanatory theories arose in the physical sciences because of their crucial importance in helping the ancient philosophers to sort out the buzzing, blooming confusion of the physical world, as they tried to assess causes and effects. Because of the success of theory in helping to predict and control physical phenomena, behavioral sciences have adopted the physical scientists' penchant for theorizing—with mixed effects.

The advantages and disadvantages of explanatory theories in the behavioral sciences differ from those in the natural sciences in quality, if not in kind. Out of the field of philosophy (and, particularly for the Western world, the type of philosophy associated with Plato and Aristotle) arose theories about the nature of humanity that still are having a profound influence on the development of theories in the behavioral sciences. Aristotle's emphasis on "either/or" logic in scientific inquiry has proved most useful in hypothesis-testing in the physical sciences and in differential diagnosis in medicine. However, an increasing number of students of the philosophy of science would agree with Skinner that there has been a "widespread reaction against the assumption that the natural sciences offer an adequate or even a relevant model for the practice of the social disciplines" (1985, p.6).

One of the reasons for skepticism about theories in the behavioral sciences is that the old-style either/or binary approach borrowed from the natural sciences often puts too much emphasis on single-cause explanations of human behavior where a broader "systems" or "multi-causal" or "field-theoretical" approach would be more appropriate (Lewin, 1936). Thus some biologists will talk as though genetic causes of alcoholism are paramount, whereas some environmental reductionists will lay the blame for alcoholism

exclusively on cultural causes. Reductionist chauvinism has plagued the field of alcoholism in particular, because of the human tendency to aggrandize the importance of one's own work, and because many scientists and practitioners lack the background requisite to broad perspectives on the complex dynamics of the various biological, personal, and environmental influences that bear on the development of alcoholism.

Adding to this recipe for wrangling or non-cooperation among the various disciplines in trying to solve the problems is the inertia of the status quo that has developed in the U.S. and Canada since considerable (though allegedly never adequate) public funds were made available for treatment of alcoholism in the early 1970s. Since that time, although there has been some success in dealing with alcohol problems (reflected in some reduction in per capita consumption of spirits, and a somewhat greater public awareness of the risks of drinking during pregnancy), the costs in lives and dollars lost because of the misuse of alcohol appear to be even greater than they were a generation ago. One of the reasons for this poor record may be that the medical-treatment and research-grant systems have put most of their emphasis upon study and treatment of *advanced* alcoholism rather than upon prevention. This has been the natural outgrowth of a political climate in which governments are reluctant to risk offending the alcohol industry — a powerful lobby and a prime source of campaign funds — by instituting increased controls (Cahalan, 1987).

This status quo is buttressed by grant and contract mechanisms, which have put a premium on rewarding the work of specialized individual scientists rather than on reinforcing the teamwork of task forces representing all of the relevant disciplines. Thus it is little wonder that researchers in the field of alcohol have been reluctant to venture forth from their safe single-discipline niches into the uncertain realm of working with alien disciplines to develop broader theories and more comprehensive methods for dealing with alcohol problems.

To sum up, *Theories on Alcoholism* can be an influence toward significant improvement in the development of theory and practice in dealing with alcohol problems if the theories described in these eleven chapters are examined critically in relation to the goal of integrating them into a more comprehensive network of interactive (as opposed to unidirectional) systems-oriented cause/effect explanations for human behavior. Enough is now known about the multi-causal nature of alcohol problems to establish where there is greater profit to be gained: by focusing on how the various theories on alcoholism can be integrated, rather than expending additional effort advocating the primacy of any single theory. A thorough study of the following chapters also can yield better insights into theory and practice in other fields with addiction components, such as addictions other than to alcohol (e.g., smoking), gambling, and other behaviors that have such powerful short-term reinforcements and such disastrous long-term consequences.

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INTRODUCTION

C. Douglas Chaudron and D. Adrian Wilkinson

RATIONALE FOR THE BOOK

The impetus for this project came to us from three sources. We both teach about addictions, and we have found that our students regularly request theoretical explanations of addiction or of alcoholism. They challenge us to review the limits of, and empirical support for, the theories that we discuss. Persons receiving treatment frequently make the same request, and both groups do so in apparent confidence of an authoritative reply. When one responds to such questions in terms of the complexity, multifactorial determination, and definitional ambiguity of the construct, a handy reference is often requested; we have been unable to supply one that would satisfy the eager learner. Clearly, authoritative reference texts (e.g., Pattison & Kaufman, 1982) and series (e.g., Kissin & Begleiter, 1971-1983; Galanter, 1983-1987; Gibbins et al., 1974-1986) exist, but these do not explicitly address the wish to compare among theoretical approaches to alcoholism. Some editions address theories of alcoholism, but from the standpoint of one discipline such as psychology (e.g., Blane & Leonard, 1987; Hodgson, 1987; Peele, 1987), and as such do not intend the scope for which we hope. Hence, we perceived the need for a broad-based, explicitly theoretical volume on alcoholism, not as a lay guide but as a solid reference for students interested in the variety, scope, and formal properties of the theories.

A second group to whom we hope the volume will be useful are persons involved in the development of policies aimed at minimizing the detrimental effects of alcoholism and associated problems. In the context of current concerns about the allocation of public and private resources towards these ends (Gordis, 1987), one hopes that policymakers and their advisers would know what phenomena theories deal with, and how secure are the grounds for theory-based proposals.

Our own priorities also pressured us to embark on the project. We have both been interested students of the addictions for more than a decade, and, coming as we both do from backgrounds in philosophy and science, we have been preoccupied with issues in the philosophy of science as they pertain to addiction studies. As this volume indicates, there is a range of scientific theories that pretend to relevance in explaining alcoholism. It seemed to us that none of them would claim to be comprehensive, or general, theories of alcoholism, though all would claim some bearing on alcoholism. This state of affairs might relate to the variety of meanings of the alleged scientific construct *alcoholism*, or alternatively to various properties that alcoholism might have (e.g., prevalence, incidence, etiology, response to treatment), each of which might require a different theoretical explanation. We believed that it would be

useful to persuade acknowledged experts in the field to outline the formal properties of the theory they were evaluating, as it applied to alcoholism, to indicate the limits of the phenomena of alcoholism that the particular theory claimed to explain, and the tests that were considered adequate in evaluating the theoretical position. The resulting volume seems to us to have vindicated our belief. Though the data reviewed here can be found elsewhere, the attempt to integrate them in a common expository framework is unique. We think this common framework will help to clarify where there is true theoretical disagreement, where different phenomena are being explained, and where the level of explanation of the same phenomenon varies.

Critics might decry the use in our title of what some consider an outmoded and problematic term — *alcoholism* (Edwards, 1987). Others might argue that a theoretical book should tackle not the arbitrarily restricted issue of alcoholism but the phenomenon of which it is but an instance — *addiction*.

In developing our plan for this volume, we did consider embracing the addictions generally. There were two major impediments to such a strategy. Firstly, although the definition of *alcoholism* is problematic, there is at least a broad consensus that it relates directly to the heavy consumption of alcohol. In the case of the addictions there is no such consensus (Orford, 1985). Some behaviors considered to be addictive do not obviously involve consummatory acts (e.g., gambling, hypersexuality) or do not obviously involve excess (e.g., anorexia). In short, the boundary conditions of the set of phenomena termed *addictive behaviors* have not been well enough defined for our purposes.

The second impediment to a volume on the addictions was much more practical. Scholars in our field tend to specialize in the study of a specific addiction rather than the range of them. Alcoholism is probably the most intensely studied, and, for some of the theoretical approaches we hoped to cover, almost all of the work has been on alcohol. Hence, we were most likely to find the range of authors to which we aspired by restricting our book to alcohol. Furthermore, we reasoned that if alcoholism is a *bona fide* addiction, then the elucidation of theories on alcoholism should illuminate the field of addictions generally. Indeed we wondered whether, in the section "Boundaries of the Theory," our authors would deal with the issue of generalization to other addictions. This was not to be, but it remains for us an interesting issue for the exploration of which we believe the chapters assembled here can form a useful starting point.

A variety of questions could be asked concerning the chapters, such as: To what extent can the different theories account for the development of this addiction rather than that, or both this and that? Does Theory X enable us to understand the great variations in *abuse liability* or *dependence potential* in the various drugs of abuse? Does Theory X inform us of the relevance of the constructs *abuse liability* and *dependence potential* to hypersexuality, gambling, and eating disorders — all acknowledged addictive behaviors? At the purely conceptual level, how distorted would the theoretical constructs be if one substituted *tobacco dependence* and *smoking* for *alcoholism* and *alcohol consumption* in all of the theoretical sections of this book?

The term *alcoholism* is now eschewed by many authorities in the field,

and with considerable justification (Edwards, 1987). We could be facetious and say that the book has been so long in production that refinements in nomenclature have simply overtaken it and made our title an anachronism before publication; but this would not explain our sticking with it. The reality is that alternatives were not readily to hand. This is not a book of theories on alcohol dependence, or of theories on alcohol use or abuse, though all of these areas are touched upon in some of the chapters. In an editorial entitled "No 'Alcoholism' Please, We're British," Edwards (1987) asserts, with his usual pithiness, "At one level the challenge to the word 'alcoholism' is a complaint against the reification of diverse and multiply-determined behaviours as one entity" (p. 1060). In planning this volume we intended just such a complaint. Paradoxically, however, we ultimately decided that, in communicating to potential readers what the book was about, we could not improve upon our original selection of title.

THE SELECTION OF THEORIES

How does one determine the nature and number of theories on alcoholism to be represented in a volume with the pretensions of the present one? This question has no definitive answer, but it is a fair question, and we attempt a response by considering some notable absentees from our list. Explanations of their absence will clarify our selection process.

Probably the most notable absentee from the list of theories in this book is the "disease concept of alcoholism," proposed by Jellinek (1960) and still very influential. Its successor — some (e.g., Shaw, 1985) would argue its most recent incarnation — the "alcohol dependence syndrome" (Edwards, 1986), is also absent. There are two reasons for these omissions. Firstly, to the extent that these are theories, we consider them to be theories *of* alcoholism rather than theories *on* alcoholism. This distinction may sound like hair splitting, but we assert that it is not. For many years now it has been generally agreed that alcoholism is a multiply determined phenomenon, or in currently fashionable terms a "biopsychosocial" entity. We were interested in exploring in the present book what slice of this biopsychosocial pie the proponents of a range of bio-, psycho-, and social perspectives were prepared to claim for their theory, and whom they viewed as misguided rivals and whom as legitimate claimants to other sections of the pie.

In contrast, we view the disease concept of alcoholism and the formulation of the alcohol dependence syndrome as efforts at integration, or arguments for a biopsychosocial perspective. Furthermore, we would argue (though we will not attempt a defence of the position here) that neither of these formulations represents nor was ever intended to represent a *theory* of alcoholism. They seem to us to be the equivalent of taxonomic systems in biological studies — which serve an essential descriptive scientific purpose, but do not represent theories of biological functions. In this light, the disease concept of alcoholism seems to us an organizing descriptive framework. This framework gave rise to some specific explanatory hypotheses that Jellinek advanced as such; for example, the construct of "loss of control" has been the object of empirical inquiry from the perspective of various theories (see, e.g.,

the chapters by Tabakoff and Hoffman and by Wilson, this volume). In short, though we applaud with others these pioneering *descriptive* scientific advances in the field of addiction studies, we do not see them as constituting specifically *explanatory* contributions.

Even if readers accept the above arguments for exclusion, there are a number of potential theories on alcoholism that might have been included and were not. A recent book, *Theories on Drug Abuse: Selected Contemporary Perspectives* (Lettieri, Sayers, & Pearson, 1980), contained 43 contributions, and such a number would have been impossible within the present framework. However, there are certain candidates whose absence might clearly raise questions.

There is no chapter on operant conditioning, though much pioneering research on alcohol use has been conducted within this framework (Mello & Mendelson, 1987). In our original plan such a chapter was included, but the author withdrew. However, since we had a chapter on social learning theory, which purports to embrace all conditioning phenomena, and a chapter from the respondent conditioning perspective, we decided that the omission was not crucial.

A chapter on the application of humanistic psychology was also planned. In this case we received a fine first draft, but reluctantly felt obliged not to include the contribution because of the want of empirical studies that could be brought to bear on the subject. Other perspectives, such as the existential or Marxist, would undoubtedly purport to have the capacity to offer scientific explanations of aspects of alcoholism, but our search revealed no relevant literature or scholars prepared to undertake the task.

Finally, it is frequently asserted by some persons treating clients with alcohol problems that the spiritual state of the client should be evaluated. This perspective presumably implies that spirituality is judged to have an important bearing on the condition. However, we encountered no formal theoretical statement of this position. Thus, for lack of an author, or lack of data, some of the positions that one might have hoped to encounter in this volume are absent. We hope that the omission will spur adherents to contribute their positions to the literature.

The list of chapters with which we have wound up must inevitably reflect our biases, but we believe that it is reasonably comprehensive. As mentioned above, alcoholism is now often touted as a biopsychosocial entity, and the range of the chapters reflects that view. We are confident that if a content analysis of alcohol studies could be undertaken, there would be few that would not be directly related to at least one of the positions here. That is the state of affairs we aimed at in planning the book.

When we conceived of this volume, one of our editorial aims was to achieve sufficient homogeneity of format and style in the chapters to permit ready comparisons among the theoretical positions represented. To increase the chances of succeeding, we sent detailed prescriptions to the authors along with invitations to contribute. We would like here to acknowledge their remarkable collaboration with our request. This aim for uniformity of organization and style led to the editorial work being more protracted than is usual,

and this factor (as well as others beyond our control) contributed to the length of the process. Hence, an objective of this introduction is to express our sincere gratitude to our authors for their forbearance in awaiting publication, particularly those who responded promptly and with scrupulous care to the detailed instructions we sent them.

We expected that one uniformity in the chapters would be their coming from the vantage of a critical protagonist, and indeed the contributors were invited because we judged each to have the abilities for that task. All but one of the authors responded in this way. The exception is Lester, whose chapter is very critical of the genetic theory that has found widespread favor during the last decade (Desmond, 1987). Of course this has meant that genetic theory is not comparably presented to the others, but interested readers can readily find the protagonists' views in works cited by Lester (e.g., Goodwin, 1976; NIAAA, 1985).

We worried about this apparent imbalance in the presentation of positions represented in the book. Would the other theoretical approaches have fared any better than the genetic if subjected to the kind of fierce datum-by-datum scrutiny brought to bear by Lester? That is an empirical question to which we lack the answer. We decided to treat Lester's contribution as a welcome addition to the book.

The inclusion of his chapter, written from the vantage of a committed skeptic, creates a scenario in which each of the other contributions might have been evaluated, and therefore generates a useful alternative model of theory evaluation. We wonder with what confidence protagonists for the other theoretical positions represented here would view the data they have levied, if they had expected the kind of critical evaluation that Lester has brought to bear on his selected topic. But surely in science the data must be unimpeachable? "Impeaching" a program of research is an extremely time-consuming activity, so it is likely that only programs that achieve considerable general prominence, as genetic theory recently has in relation to alcohol problems, will seem worthy of such assault (see also the discussion of the "controlled drinking" controversy in the chapter by Wilson).

OUR CHARGE TO THE AUTHORS

We have previously mentioned our frustration in attempting to identify existing presentations of theories on alcoholism. Even where useful synopses exist, comparisons between theoretical positions are difficult because of the variety of methods by which authors have elected to lay out their positions. In some cases, they have been presented as explanatory of, say, abnormal behavior or deviance generally, with passing reference to alcoholism. Various authors have given more emphasis or less emphasis to the implications and applications of theories, to the nature of theory-relevant research, or to the need for further confirmatory studies. This variation makes intertheoretic comparisons difficult; hence our request to the authors to organize their contributions according to a common set of headings.

A section on "Principal Elements of the Theory" would introduce the theory and acquaint the reader with the phenomena that it purports to explain,

its formal structure, and the scientific concepts in which it is grounded. A succinct exposition of the theory was expected in this section.

Under the heading "Boundaries of the Theory" the authors were asked to delineate the range of alcohol-related phenomena embraced by the theory. We hoped to learn what the authors viewed as the limits of the theory *qua* theory, as distinct from empirical limits of the theory's explanatory power arising from the incompleteness of theory-driven research. For example, a certain sociological theory might apply only to populations and make no predictions about individuals (a theoretical limitation), whereas a given psychological or neurobiological theory that would generate predictions about persons might only have been tested in the rat model (an empirical limitation).

We asked the authors to include in the section on *boundaries* a contrasting characterization of those phenomena defined as lying beyond the purview of the theory. We hoped that this comparison would serve as an antidote to the tendency to think that a theory *on* alcoholism, however sound, should be treated as a theory *of* alcoholism.

The section on "Historical Development of the Theory" was intended to reveal who have been the major figures in its evolution and what extensions or restrictions of the theory have occurred. We noted that most theories on alcoholism were developed to explain other phenomena and then generalized to incorporate some aspects of alcoholism. Where this was true of a theory, we asked contributors to describe its original form, the phenomena that inspired the originator, and the reasons for incorporating alcoholism into this set of phenomena. Whatever the origins of the theory, we asked for a description of the landmark research associating it with alcoholism and the historical progress of such research.

Under "Characteristic Research Methods" we asked for a description of the basic methodology or methodologies used in elucidating the theory. In the ideal case the nature of the methods employed would bear a clear logical relationship to the structure of the theory and the predictions that derive from it. We hoped that the material in this section would allow readers to evaluate the coherence of that relationship.

"Research Results and Needs" was expected to contain a presentation of the principal current empirical support for a particular theoretical approach to alcoholism. We expressed especial interest in a discussion of any methodological problems with what are perceived as the major studies of the theory. We also asked to hear of major empirical advances that would be required to extend the theory to the limits of its explanatory power, and of technical or methodological advances that promise future progress. Our hope was that the material in this section would enable the reader to evaluate the theory in regard to its current level of empirical support and the prospects for growth in that support. Lack of accumulated empirical support was, as mentioned earlier, one of the bases for the difficult decision not to include certain theoretical perspectives, despite their intuitive appeal.

The section "Relations to Other Theories" was included to encourage the contributors to reflect upon competition and compatibility among theories, with their own topic as the reference point. Understanding what a given

theory aims to explain, and having reviewed the empirical support for its claims, a reader should be able to judge whether, among theories attempting to explain the same set of alcohol-related phenomena, one is more successful than another. On the other hand, the reader may come to see that two apparently conflicting positions are in fact attempting explanations of somewhat different sets of phenomena, with one claiming to explain a larger proportion of the variance in alcoholism. Some theoretical positions will be completely non-overlapping in the data they purport to explain.

Finally we requested a section, "Practical Implications," for each of the theoretical positions represented in the volume. Implications were bound to have been touched upon in other sections, but we thought it useful to ask each author specifically to address how the proposed theory had led to attempts to prevent or reduce alcohol problems, and also how it could be applied to these ends in the future.

CONCLUDING COMMENTS

As we indicated at the start of this introduction, the production of this book has been a protracted endeavor and must have been frustrating for our contributors. We thank them profoundly for their diligence and their patience. Have the effort and the wait been justified? We believe they have, but of course we are far from objective. A test that could be applied would be to imagine that one wished to construct a theory of alcoholism; would the chapters in this volume form a useful basis for such an effort? In constructing such a general theory, which of the ideas in this book would play a central role, and which would be on the fringes, or ignored as redundant or irrelevant? What crucial aspects of alcoholism or addiction are not touched on here? We believe the present volume could serve as the basis for such a conceptual exercise, and if so, it has succeeded.

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THEORIES on
ALCOHOLISM

1. GENETIC THEORY

An Assessment of the Heritability of Alcoholism

David Lester

1. Principal Elements of the Theory 1
2. Historical Development of the Theory 3
3. Characteristic Research Methods 4
4. Boundaries of the Theory 4
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1. PRINCIPAL ELEMENTS OF THE THEORY

Genetics deals with characters transmitted from parents to offspring; such traits are "inborn" and hereditary, not acquired, and are related to specific facets of the organism's biological structure and function. "Inborn errors of metabolism" account for a wide variety of human maladies. The goal of studies on the possible hereditary nature of alcoholism is to elucidate the biological mechanisms responsible for the development of alcohol abuse and alcoholism, and thus, as with phenylketonuria, to be in a position to provide rational counselling, prevention, or treatment methodology. My aim here is to examine, critically, the reports of those investigations that deal with this issue, focusing especially on those reports of the last several decades that have led most people to accept as proven the heritability of alcohol abuse and alcoholism.

Molecular biology has established the structural reality of the units of heredity, the genes. Because offspring share 50% of the genes of each parent, their own assemblage of genes — their genotype — is unique and not the same as that of either parent; the parent genotypes are disassembled and reconstituted in the progeny from the transmitted genes in ovum and sperm. As the organism develops prenatally and postnatally, under a variety of changing environmental circumstances, the interaction of genotype and environment produces the multitudinous characters and traits that we recognize as the individual's phenotype(s). The science of genetics deals with the transmission

and combinations of genes, first elucidated by Gregor Mendel. The expression of the genotypes as observable wrinkling of peas, blood groups, eye color, stature, butterfat yield, abdominal bristle number in *Drosophila*, and like traits has long been a staple of biology. The development of a specific genotype in varying environments (the environment being both the genetic and the nongenetic environment) can result in the production of a range of such phenotypes: the functional relation between phenotype and varying environment for a specific genotype is the norm of reaction (Lewontin, 1982).

For plants and for some species of animals, whose genotype can be isolated, propagated, and controlled, the norm of reaction is determinable: the genotype can be replicated, the environment altered, and a variety of outcomes measured. Although human genotypes in all probability exhibit such norms of reaction, no such experiment appears likely. Such an experimental program is restricted to a narrow range of genotypes and their phenotypes, to relatively simple traits in which a straightforward cause-and-effect relation can be established. For concepts like intelligence, or schizophrenia, or alcoholism, there is no evidence that simple relationships exist; indeed, there is every reason to believe that the highest levels of organic function are involved, embracing the most complex developing and evolving relationships of humans as social beings. It will be argued that to assign values to the contributions of "nature" and "nurture" appears a not particularly scientific exercise for the elucidation of such specifically human problems as alcoholism.

Falconer (1964) points out that the study of variation of metric characters (e.g., height), and the partition of this variance into components attributable to different causes, is the way genetic questions are formulated. The determination of the relative magnitude of these components is the basis for the estimation of heritability. The total or phenotypic (V_p) variability is the sum of the genotypic (V_g) and environmental (V_e) variances. The genotypic variance is divided into, and is the sum of, the additive (V_a) variance (also called the breeding value, the major component of the genotypic variance), the dominance (V_d) variance (where the effect of the gene is recognized in the zygote) and their interaction (V_i). The quotient V_a/V_p is called the heritability, the degree of resemblance between relatives. This relationship assesses the relative importance of the breeding value as a portion of the total variance of the phenotype. For nonmetric, qualitative traits, genetic involvement is assessed (e.g., in twin pairs) by concordance rates; a twin pair is usually considered concordant if either both or neither possess a particular trait and discordant if one of the pair possesses the trait and the other does not (Stern, 1960, p.554). In some cases (e.g., Kajii, 1960a), concordance has been defined as only the possession of the trait by both co-twins, excluding the case where neither possesses it.

The heritability value is of undoubted importance to the animal breeder who seeks increased milk production in cattle whose life cycle is under almost total control. Its importance for humans, subject to a variety of biological and social influences, is, at best, substantially less. There is, today, no evidence of a correspondence between human alcoholism and one gene or constellation of genes, and unless it can be shown that a significant hereditary component is

present, research investigations in this area would appear to be pursuing a scientific will-o'-the-wisp. In any case, the demonstration of a hereditary component does not, of course, indicate the specific genetic involvement: further investigation is required to elucidate the mechanism of action.

2. HISTORICAL DEVELOPMENT OF THE THEORY

An appropriate starting point for any discussion of heredity and alcoholism is the position taken by Goodwin (1982) vis-à-vis Elvin M. Jellinek's 1944 review lecture, "Heredity of the Alcoholic" (Jellinek, 1945). Goodwin contends that Jellinek was dogmatic in stating that alcoholism was transmitted socially. The dogma of the day, however, was that alcoholism was hereditary, and it was indeed challenged by Jellinek's assessment of the contemporary evidence. That the "notion that alcoholism is hereditary is old" has, in fact, been asserted by Goodwin himself (1976). It is thus not amiss to emphasize that for many decades prior to 1944 (and thereafter) the laws of the various states allowed the compulsory sterilization of those considered genetically degenerate, including (in addition to the insane, the feeble-minded, moral and sexual perverts, habitual criminals, and rapists) drunkards, epileptics, and syphilitics, and that such laws carried (in an 8 to 1 decision) the 1927 legal imprimatur of the Supreme Court of the United States (Buck v. Bell, Superintendent; Paul, 1973). For Jellinek to conclude that "no evidence has ever been adduced to show that this heredity, by *necessity* [my emphasis], had to express itself in inebriety" was to judge the evidence fairly, and he continued by stating, "The only permissible conclusion is that not a disposition toward alcoholism is inherited but rather a constitution involving such instability as does not offer sufficient resistance to the social risks of inebriety. The inherited constitution is merely a suitable breeding ground for inebriety" (p.109).

Careful study of Jellinek's 1944 lecture should convince readers that the admittedly undogmatic Jellinek believed firmly that no matter what weight was assigned to hereditary factors, the mental hygienist was, in fact, "not faced by implacable fate" (p.110). Having worked for many years as a statistician concerned with plant breeding, Jellinek was well aware that even a perfectly heritable trait could be substantially changed by environment. How tangled the web in which, on the one hand, the creator of the "disease concept" of alcoholism (Jellinek, 1960) is criticized as a dogged opponent of the hereditary nature of alcoholism (Goodwin, 1982), and yet, on the other hand, Peele (1984), who favors a social explanation, notes:

The disease theory of alcoholism has the merit of bringing troubled people into the care of hospitals and doctors, an advantage appreciated particularly by physicians themselves, who tend to see human problems in terms of the medical model: disease, treatment, cure. Yet it posits an inborn organic cause, a bodily deficiency, where there may be none, and for this reason the theory is troubling. Alcoholism may at its roots be a social and cultural problem, not a medical one. (pp.15-16)

It is sadly ironic how Jellinek's views on alcoholism and heredity have

been misinterpreted — else how explain the serious suggestion by an adherent of all-encompassing hereditary doctrine that alcoholism be renamed Jellinek's Disease (Fitzgerald, 1983)?

3. CHARACTERISTIC RESEARCH METHODS

The methodology used in research on the heritability of alcoholism is characteristic of most investigations dealing with complex traits. None of the methods employed are original in the sense that they have been developed for the study of alcoholism, nor are they applied in a manner fundamentally different from that in which they have been applied to the study of other human attributes, such as schizophrenia. Because the definitions of complex traits such as alcoholism can be narrow or broad, the definition to be employed is of cardinal importance: how else recognize the appearance of the trait?

Breeding as a possible methodology is excluded on ethical grounds, as it is not for infra-human animals; but even if an animal model of alcoholism were possible (Lester, 1982), its inadequacies make it of doubtful relevance. Other methodologies are discussed in later sections of this chapter, and deal with the specific manner in which the actions of the genes are purportedly separated from the actions of their environments: comparison of identical and nonidentical twins, of half siblings raised with and without their biological parents, or of adoptees separated from their biological parents shortly after birth; the association of "alcoholism" with characters known to be genetically transmitted (markers); and the presumed correlation of heredity and elimination (metabolism) of alcohol.

Many proponents of the view that alcoholism is heritable are aware that alcohol use, and most especially alcohol abuse, introduces the special methodological problem of assessing the consequences of its antecedent chronic use upon preceding measurable attributes; there seems to be no manipulation, other than a prospective study, that avoids this pitfall. To employ an acceptable definition of alcoholism, to mount an adequately sized prospective study in which the data are generated by the study itself (rather than by uncontrolled outside agencies), and to evaluate the weight to be given to the genes (by methodology akin to that of Cloninger et al., 1981) is a formidable task. The risk in such prospective studies is that there is never a guarantee that all the right questions (Regier et al., 1984; Robins et al., 1984) will have been asked, let alone that significant answers will be found; but if alcoholism is to any extent heritable in any context, then such a study should be the next one pursued.

4. BOUNDARIES OF THE THEORY

Given a demonstration of the weight to be given to the "heritability" of alcoholism, some important limitations of such a finding would still have to be borne in mind. As Lewontin et al. (1984) state (anent IQ):

The heritability of a trait only gives information about how much genetic and environmental variation exists in the population *in the current set of environments* [emphasis in the original]. It has absolutely no predictive power for the result of changing the set of environments...

The confusion of "heritable" with "unchangeable" is part of a general misconception about genes and development. The phenotype of an organism is changing and developing at all times. Some changes are irreversible and some reversible, but these categories cross those of heritable and nonheritable. The loss of an eye, an arm, or a leg is irreversible but not heritable. The appearance of Wilson's disease [a defect of copper metabolism inherited as a single gene and curable with penicillamine] is heritable but not irreversible. The morphological defect that causes blue babies is congenital, nonheritable, irreversible under normal developmental conditions, but reversible surgically. The extent to which morphological, physiological and mental characteristics do or do not change in the course of individual lifetimes and the history of the species is a matter of historical contingency only. The variation from person to person in the ability to do arithmetic, whatever its source, is trivial compared to the immense increase in calculating power that has been put into the hands of even the poorest student of mathematics by the pocket electronic calculator. The best studies in the world of the heritability of arithmetic skill could not have predicted that historical change. (pp.116-117)

Placing a value on the heritability of alcoholism, which is assuredly far less than 1.0, tells us nothing about the biochemical, morphological, physiological, or other biological substrata that may be necessary if alcohol abuse or alcoholism is to be manifest, let alone the variety of social, cultural, economic, historical, and other environmental factors that may be, and in all probability are, involved.

The determination of the heritability of alcoholism has, of course, been attempted on many occasions, and it is of obvious interest to examine the results. My present assessment of the studies of the last 40 years leads me to conclude that little change is required in the 1944 judgment of Jellinek. Previous reviews of the heredity of alcoholism have in large part been uncritical, but several excellent reviews on different aspects of this topic have been recently published (Cabaniss, 1979; Murray et al., 1983).

5. RESEARCH RESULTS AND NEEDS

I hope here to extend the critiques of Cabaniss (1979) and Murray et al. (1983); these authors, in lengthy reviews, have examined the available twin, adoption, and half sibling studies, indicating their many methodological and other deficiencies, and reaching essentially the same conclusions. Cabaniss concludes (p.61) that "the evidence for a genetic factor in alcoholism which acts selectively on some individuals and not on others is less than adequate," while Murray et al. conclude that there may be a "modest genetic effect on both normal drinking and alcoholism in men, though similar evidence for women is so far lacking" (p.25). Many of the deficiencies noted by these critics overlap, and many do not; and many other serious deficiencies have been overlooked by

them and will be examined in what follows. I restrict my critique to those well-known reports that allowed evaluation of the study's conclusions because they employed adequate numbers of subjects, applied appropriate definitions of alcohol abuse and alcoholism, and provided a sufficiency of data for analysis.

5.1 TWIN STUDIES

Twin studies compare monozygotic (MZ: arising from a single ovum and sperm) and dizygotic (DZ: arising from two separate ova and sperm) twins, the MZ twins with identical genotypes and the DZ twins (same-age siblings) sharing only half their genes. DZ twins may thus be fraternal, sororal, or of unlike sex. Except as specifically noted, most of the studies have employed male twin pairs. For any qualitative character or trait, greater concordance (similarity) between MZ pairs as compared to DZ pairs is taken to result from the MZs' genetic identity, and the ratio of their concordances reflects the degree of genetic input; any discordance in an MZ pair is assumed to be the result of environmental differences acting on an identical genotype.

Twin studies depend on various assumptions, some of questionable validity. They assume that mating of the parents has been random (i.e., that there is no conscious preference for marriage partners with similar obvious traits), that no dominance or other genetic effects are involved in the particular trait (the trait may arise from asymmetry of alleles at a locus or loci), and that within-pair environmental variance is the same in DZ as in MZ twins. The degree of assortative (nonrandom) mating among alcoholics is not known, but it is known to be high for traits such as height and body size. It is almost certainly the case that DZ twins are exposed to more dissimilar environments than MZ twins. Indeed, this difference is obviously true from conception onward: DZ twins generally develop in separate placentas. Postnatally the environments diverge even more. MZ twins are frequently confused with one another and treated as the same person. Because they share fewer traits, DZ twins are not only more likely to be differentiated by others but also more likely to differ in their talents, interests, friends, and occupations, and share far fewer common experiences; their greater genetic dissimilarity ensures that the sets of environments that they experience will be more dissimilar than the sets encountered by MZ pairs. The fact that intra-pair environmental variances are unequal means that the ostensible heritability calculated will perforce include these unequal variances.

In addition to this basic conceptual deficiency, there is the possibility of misclassification of MZ and DZ pairs. Other limitations are discussed by Stern (1960).

In the first study using twins, Kaij (1960a,b) drew his sample of 48 MZ and 126 DZ twin pairs from all male sets of twins born between 1890 and 1939 in Skane (southern Sweden) in whom at least one of each pair (an index twin, or proband) had at some time been registered with either of the region's two County Temperance Boards. (The MZ twins constituted 27.6% of the sample, a percentage that appears to be representative of all twins born: in the period 1941–1960, for example, the percentage of MZ pairs born in Sweden was 24.6%, according to the National Center for Health Statistics.) Each of the

twins was categorized as to drinking on the basis of official register information on a scale from *a* to *e*, and also categorized with information obtained at their interview with Kaij on a scale from 0 (abstainer) to 4 (chronic alcoholic); the latter classification was not made on about 10% of the pairs, so that only official register data were used. To the best of my knowledge, if the co-twin did not appear in the Temperance Boards' registers, there could be no "official register" information; but Kaij does not enlighten the reader how he obtained information about drinking habits in the absence of registration of the co-twin. I assume that, on the basis of the interview with the proband (or with another informant), Kaij himself decided how to categorize the co-twin, and thus introduced risk of further bias.

Those abusers of alcohol who came to the attention of the Temperance Boards are those who made a public display of their abuse, whereas other, more private, abusers were excluded. Cabaniss thus finds herself in agreement with Kaij that the sample of abusers, although representative of those registered with the Boards, are not representative of alcohol abusers and may be, admittedly, psychopaths and not primary alcoholics. Cabaniss points out that of 74 MZ individuals, only 17 were adjudged "normal and healthy." Moreover, interviewing was not blind: Kaij might well have more often assigned twins looking alike to the same class than he would those looking more unlike.

Murray et al. (1983) indicate that the element of uncertainty with regard to the zygosity of the twin pairs employed may be higher than expected, pointing (probably inaccurately) to the low proportion of MZ twins in the sample. Kaij, unable to obtain blood groups on all pairs, states that "a few MZ pairs may be concealed in the DZ group," which acknowledgment Murray et al. believe "raises the question of whether MZ twins discordant for drinking, who often look very different, may have been wrongly considered DZ" (p.29). Such misclassification would increase the concordance rate for MZ. Obtaining information from relatives and nonaffected MZ co-twins may lead to a denial of monozygosity, inflating concordance for MZ and discordance for DZ pairs. With regard to the representativeness of the sample, Murray et al. make the further criticism that there was a much higher rate of alcohol-related criminal acts in the Kaij sample than in most samples of alcoholics. These authors then consider the problem posed by the fact that DZ twins were interviewed only once, for no longer than 60 minutes, whereas most of the MZ pairs received a far lengthier and detailed psychometric examination in a university setting: more information was thus elicited about the drinking habits of MZ than DZ pairs.

While the classification based on official records indicated that 25.4% (15 of 59) of the MZ pairs and 15.8% (23 of 146) of the DZ pairs fell into the same drinking class (with $\chi^2 = 2.0012, p = .157$), Kaij's own additional "compound" classification yielded a concordance of 53.5% (31 of 58) for MZ and 28.3% (39 of 138) for DZ pairs ($p < .0014$). But as Murray et al. point out, "co-twins less adequately investigated might have appeared wrongly discordant, thus lowering the DZ concordance rates" (pp.29-30) and inflating the calculated contribution of heredity.

A major point made by Cabaniss relates to the definition of concordance employed by Kaij. She points out that the differences in concordance obtained

by Kaij from his Table 12 (and shown in Kaij's Table 13) are calculated from pairs where both possessed the trait (from any of the classes), with $\chi^2 = 10.21$ ($p < .0015$). Cabaniss recalculated the differences for each of Kaij's classes 1 to 4 (using the data for *all* twin pairs), defining concordance as 100% minus the percentage discordant for that class, and for none of the four classes are the concordances of MZ pairs significantly higher than for DZ pairs (Cabaniss: Table 2, p.22), there being, under this traditional definition, a concordance of 77.6, 65.5, 79.3, and 89.7% ($n = 58$) for MZ twins in classes 1 to 4, respectively, versus 58.7, 60.9, 73.2, and 81.9% among DZ twins. The legitimacy of this argument may be questioned because of the large number of pairs in which neither member of a pair possessed the trait denominating one of the four categories. But it is unarguable that if any one of the four classes is analysed separately, with Kaij's definition of concordance, only class 2 shows a significant difference in concordance between MZ and DZ pairs.

Neither Cabaniss nor Murray et al. noticed other defects in Kaij's study. For example, MZ pair #47 adjudged concordant (both officially *a*) is described in this manner:

The information about this pair is derived from a 13-year younger brother, who saw the author [Kaij] on account of heavy alcohol abuse. He said his parents had died when he was young, and he did not know much about them. The twins were said to have been very much alike and often mistaken for each other. Both went to sea in early years, and the informant had not seen much of them. One of them was killed in a shipwreck at age 20. The other is still at sea. He was convicted at age 23 (then proband) and at ages 24, 30 and 33. (Kaij, 1960a, p.118)

So much for monozygosity! At least one of the tables (Table 12) contains information at variance with Table 26: Table 12 shows four pairs of MZ twins at a concordant class 1, but Table 26 shows six — and so on! There are many other inconsistencies within and between tables that do not enhance confidence in Kaij's conclusions.

The reader may have noticed that Cabaniss's calculations, and those of Murray et al., refer to 59 MZ and 146 DZ pairs; yet Kaij chose only 48 and 126 such pairs. Actually Kaij found 64 probands among MZ twins and 150 probands among DZ twins; but the 64 MZ probands "were members of 48 twin pairs" and the 150 DZ probands were members of 126 DZ pairs. Nine pairs (five MZ and four DZ) were eliminated, leaving 59 MZ and 146 DZ. Astonishingly, neither Cabaniss nor Murray et al. comment on this method of counting twin pairs. Kaij justifies this increase (48 to 59 and 126 to 146) by stating: "When comparisons of *intrapair* correlations of drinking habits between DZ and MZ pairs are made, Weinberg's so-called 'proband method' was used, which means that when both members of a pair are probands the pair is counted twice" (p.30). No reference is given for Weinberg, but it is undoubtedly to the method proposed by Weinberg (1912, 1928) and misapplied by Kaij. (Weinberg, later others, proposed a method for rectifying incomplete sibling data when calculating the frequencies of a recessive trait; see, for example, the discussion by Bailey, 1951, p.216.) The event, therefore, of appearance on the register of the Temperance Boards allows some pairs to be counted more than once: Kaij's Table 26

indicates that 16 of the 48 MZ pairs were counted twice; similar data on DZ pairs are not supplied. The major effect may be inflation of the concordance rate for MZ pairs.

Table 26 allows identification of those MZ pairs who were counted twice. Murray et al. are able to point out that "71.4% of the 14 co-twins of MZ probands were also chronic alcoholics [by Kaij's own classification], compared to 32.3% of co-twins of DZ probands" (p.28), to yield $\chi^2 = 4.5117 (p < .034)$. If Table 12 is reconstituted with the data of Table 26, then there are only six (not 10) MZ pairs concordant for class 4 out of 10 such pairs, and the 71.4% is lowered to 60%: the resultant χ^2 (for chronic alcoholism, based on six concordant and four discordant MZ pairs, and 10 concordant and 21 discordant DZ pairs) is now 1.4186 ($p < .234$). This latter calculation is, of course, also suspect because Kaij provides no specific information by which to determine which of the DZ pairs were counted twice.

I can only conclude that Kaij's study is so flawed that no conclusion, least of all about the hereditary nature of alcoholism, can be drawn. This lengthy examination has served the purpose, I hope, of demanding the closest scrutiny to such studies, made possible, of course, by the indefatigability of Lennart Kaij.

In one of the largest studies, Partanen et al. (1966) examined 902 male twin pairs chosen from all twins born in Finland between 1920 and 1929 and registered with the Finnish Central Statistical Office; a total of 641 DZ and 198 MZ twin pairs, and 63 twin pairs of undetermined zygosity, were studied. The zygosity of each pair was determined by the similarity of a variety of measures, including stature, eye and hair color, and blood typing. DZ twins comprised pairs discordant for blood type(s) or with physical attributes incompatible with MZ status. Where blood samples were unobtainable, close physical similarities alone decided the choice for MZ placement.

For six measures connected with the drinking of alcoholic beverages — density (frequency of alcohol consumption), amount (consumption per drinking session), loss of control (ability to abstain from drinking), drunkenness arrests, addictive symptoms, and social complications — only the heritability values for density and amount (0.39 and 0.36) suggested an influence of heredity (as did the values for drinking coffee or smoking tobacco). The other measures, which would also appear to reflect alcohol abuse and to be correlated with density and amount, had nonsignificant values for heritability. The tenuous nature of the apparently significant heritability values is seen in the loss-of-control measure: younger twins gave a value of 0.54, but the value for older twins was -0.07, interpretable as the cumulative effect of the environment, and paradoxical too because in the age range used older twins might have been expected to exhibit more fully developed signs of alcohol abuse. The disparity between the values for density and amount versus those for the other measures suggests that we lack some understanding of the meaning of these measures or that sampling error has produced some significant result. Because the sample contained few heavy drinkers or alcoholics, indicants such as hospitalization or liver cirrhosis were not used.

Various other twin studies reviewed by Cabaniss and Murray et al. add

little to the present discussion. However, Murray et al. report in a preliminary manner on their own study, which had (at the time of their review) examined 56 pairs of twins, in which zygosity was established, for their concordance rates for "dependence on alcohol." They found 21% of the MZ and 25% of the DZ twins concordant for this trait, an obviously nonsignificant difference, albeit weighted towards a negative heritability.

The differences in intra-pair environmental variances would be reduced if twins raised apart from an early age were to be studied, but few such pairs exist, and the few in which drinking habits are known (Kajii, 1960a,b; Newman et al., 1937; Shields, 1962) were not actually raised completely apart: in every instance the co-twins had knowledge of each other.

5.2 HALF SIBLING STUDIES

In three papers, Schuckit et al. (1972a,b,c) reported upon various stages of their investigation of a group of alcoholic probands and their half siblings. These individuals have differing genetic and environmental relatedness depending on their parentage, and thus offer a means of evaluating the contribution of heredity. The 69 probands were chosen from patients admitted to the alcohol unit of the Malcolm Bliss Mental Health Center and from the Renard Hospital (both in St. Louis, Missouri) on the basis of "severe life problems as a consequence of alcohol abuse" (1972b, p.165) defined as alcoholism. The consequence of alcohol abuse was the presence of (i) a divorce or legal separation, or (ii) a job loss, or (iii) two or more nontraffic arrests, or (iv) a hospitalization for a physical or psychiatric consequence of excessive drinking other than the index admission. Persons with any preexisting psychiatric disorder were excluded. In addition to meeting one of the mentioned criteria, the probands also had to have half siblings: 164 were found. From the probands, and 90 of their relatives, was obtained a "history of psychiatric illness in their biologic parents, in all parent figures, and in full siblings and half siblings, as well as in biologic parents and parent figures of the half siblings" (1972c, p.1133).

The half siblings can be divided into four groups (Table 3; Schuckit et al., 1972c) on the basis of whether there was an alcoholic biologic parent or not and whether the half sibling was raised with or without such a figure. Of the 46 with an alcoholic biologic parent, 24 were raised with that parent and of these 11 were alcoholics; of the 22 raised without that parent, 11 were also alcoholics. Of the 118 without an alcoholic biologic parent, 14 (of whom two were alcoholics) were raised with an alcoholic parent figure and 104 (of whom eight were alcoholics) were raised without such a figure. There were thus 32 alcoholics among the 164 half siblings, 22 of whom had an alcoholic biologic parent; and of the 132 nonalcoholic half siblings, 24 had such a parent. Thus, 69% of the alcoholic half siblings had an alcoholic biologic parent compared to only 18% of the nonalcoholic half siblings, a ratio of almost 4 ($p < .0005$). Either this table or the preceding Table 2 (or both) are in error, Table 2 showing 20 (62%) of the 32 alcoholic half siblings and 26 (20%) of the 132 nonalcoholic half siblings to have an alcoholic parent, but Table 3 giving these numbers as 22 and 24. At an earlier stage of the study (Schuckit et al., 1972a), a similar Table 2

seems to contain a similar type of error: according to the table, 61% of 23 ($n = 14$) alcoholic half siblings and 19% of 75 ($n = 14$) nonalcoholic half siblings had an alcoholic biologic parent, from which $\chi^2 = 16.141$ is calculated for the comparison; yet such numbers (14 and 9 versus 14 and 61) would yield $\chi^2 = 13.3635$; if the percentage of alcoholic biologic parents were 65% (15 rather than 14 parents), not 61%, then indeed $\chi^2 = 16.141$. These errors raise doubts about the care the authors gave to other, more critical, parts of the study. Further, even if the values favoring a greater hereditary involvement (from Table 3) are used, and a comparison made between the numbers of alcoholic and nonalcoholic half siblings with and without an alcoholic biologic parent, but raised with such a parent figure, the comparison is between 11 and 13 of the former and 2 and 12 of the latter: this gives $\chi^2 = 2.6339$ ($p = .105$). This particular comparison highlights the thinness of the results and indicates the importance to be given the authors' own rather curiously obscure caveats, as when they state:

The limitations of this type of study must be kept in mind. It is possible that a nonalcoholic biologic parent determines in some subtle way the development of alcoholism in children raised by neither biologic parent. (1972c, p.1135)

Cabaniss deplores the incomplete, and arbitrary, decision of the authors to place half siblings in either the group "raised with" an alcoholic parent figure or "raised without": in the former group were half siblings who resided a minimum of 6 years (before the age of 17), and the latter group contained those residing for less than 6 years. She suggests that living with an alcoholic parent figure for a substantial period of time (i.e., just short of 6 years) could have affected the development of alcoholism, and that a more decisive division between those half siblings who never lived and those who lived throughout the childhood years would have provided a more suitable comparison. Schuckit et al. note that the time interval was chosen not only to provide a substantial time of exposure to the parent but also to yield groups of sufficient size; they also indicate that an attempt to consider this effect yielded such small numbers in any category that "a larger sample would be needed to provide a definitive answer" (Schuckit et al., 1972c, p.1134).

Murray et al. (1983), while praising the methodological strategy, believe that the results might have been related to the 81% of the alcoholic half siblings who lived in a broken home. They also consider it "strange" that there was a lesser likelihood to become an alcoholic in those half siblings raised with the alcoholic proband. And finally, they believe the definition of alcoholism used was so broad as to mischaracterize the study as one concerned with the genetics of alcoholism, and would rather speak of it as dealing with alcohol abuse and problem drinking.

The answer to the deficiencies noted by Cabaniss, Murray et al., and not least by Schuckit et al., in addition to those already given, appears also to lie in carefully selecting a large group of probands each matched (at random) with a single same-sex half sibling. Such an approach, though it makes for the exclusion of many possible probands, and thus the achievement of a subject pool more difficult, has the decided advantage of providing a justifiably better answer and avoiding the criticisms now levelled.

5.3 ADOPTION STUDIES

Following the lecture of Jellinek on "Heredity of the Alcoholic" (1945, Lecture 9) came the lecture of Anne Roe on "Children of Alcoholic Parents Raised in Foster Homes," a report of a study of adopted children of alcoholics (Roe, 1945). Not only did it precede by more than 20 years the wider recognition of this particular strategy for genetic research in behavior, but also Jellinek was obviously well aware of Roe's work before its presentation and it influenced his own critique of prior work. (Both Jellinek and Roe were associates in what was to become the Center of Alcohol Studies at Yale University.) In contrast to the work criticized by Jellinek, Roe concluded "that the reported high incidence of alcoholism and psychosis in the offspring of alcoholics is not explicable on the basis of any hereditary factor" (p.125). However, Roe's work can be, and has been, faulted on several grounds, including the use of adoption records for the classification of the parents, the imprecision of the definition of alcoholism, and the differences between the probands and the controls as to sex, age at placement in a foster home, and so on.

Current interest in the possible hereditary nature of alcoholism began with the study by Goodwin et al. (1973) of male adoptees with a biological parent who had been hospitalized primarily for alcoholism. These "index" adoptees were chosen from among all those adopted outside their own families ($N = 5,483$) in Copenhagen and its immediate environs from 1924 to 1947, a pool established for the study of schizophrenia by Kety and others. The criteria for an index case included: alcoholic parentage (85% were the biologic father), adoption by nonrelatives before the seventh week of life, maleness, and no known contact with biological relatives. A total of 67 adoptees were selected. Two control groups were chosen, the first consisting of 70 adoptees meeting the index criteria (matched for age, sex, and time of adoption), except that neither biologic parent had a hospital record of alcohol abuse or alcoholism or other psychiatric illness, and the second consisting of 37 adoptees who "had a biological parent hospitalized for a psychiatric condition *other* than alcoholism" (p.238). The number of adoptees thus totalled 174: 67 index cases, and 70 and 37 control adoptees.

Of the sample of 174, 41 (14 probands) could not be found or refused their participation. Another adoptee was eliminated because it was not known which of two possible biologic fathers was the real one: whether this adoptee originally belonged to the index or the control group is not stated. The subjects were interviewed (in Danish, later translated into English) by a psychiatrist (without knowledge of their proband or control status). Information about demographic matters, parents, drinking practices, life style, psychopathology, etc. was secured in a structured 2-to-4-hour interview. Since there were no differences between the control groups, they were combined. This process would seem to leave a total of 132 adoptees (174 minus 41 minus 1), but the authors' arithmetic gives 133. Similarly, one would expect to have 53 (or possibly 52) probands (67 minus 14, or 67 minus 14 minus 1), but the authors calculate 55 probands; and where one might expect 80 (or possibly 79) controls (70 plus 37 minus 27, or 70 plus 37 minus 27 minus 1), the authors give the total as 78. These unexplained arithmetical inconsistencies, as well as others to be

discussed, raise questions about the care with which the study was conducted.

The adoptees were placed in one of four drinking categories (there were no teetotallers among them): moderate, heavy, problem drinker, and alcoholic. Problem drinkers and alcoholics met the criteria for a heavy drinker, with the former having insufficient problems from drinking to be alcoholic, the latter having problems from three of four areas: social disapproval of immediate family, including marital problems; job or police troubles from drinking; frequent blackouts and other consequences of intoxication and withdrawal from alcohol; and loss of control or morning drinking. In addition to their being placed in one of these four groups, alcohol's effects and consequences (e.g., hallucinations, morning drinking, drunken driving arrests, hospitalization for drinking) and the extent of psychopathology (e.g., anxiety neurosis, depression, psychiatric treatment) were noted and comparisons made between probands and controls.

The statistical tests employed were chi square (two-tailed, with Yates's correction) and appropriate Student's *t* test. In the evaluation of the chi square statistic, the use of the two-tailed probability implies no *a priori* knowledge that, for example, probands (or controls) are more (or less) likely to manifest a particular symptom or to be categorized as a moderate, heavy, or problem drinker, or as an alcoholic. The one-tailed probability is one-half of the two-tailed probability, but it demands, for the validity of its use, that any discrepancy in a direction opposite to that noted be unrelated to factors relevant to the study (Bliss, 1967; Fisher, 1954). Thus the assumption of the authors is clear: the direction of expected frequencies of the enumerations employed was not known for the two groups of adoptees. Although the authors' calculations of chi square are mostly correct, it is not always possible to gauge how they arrived at their inconsistent estimates of the probability. In some instances, Yates's correction is not used; in other instances, the one-tailed probability is used. Such usages obviously either increase the calculated chi square, thereby lowering the probability, or lower the calculated *p* directly: the result is to enhance, falsely, the plausibility of the authors' arguments for the heritability of alcoholism.

Where the data are available, I have recalculated the values of chi square and the probability associated with it. For example, Table 3 (Goodwin et al., 1973, p.240) shows that 22 (40%) probands and 19 (24%) controls required some psychiatric treatment, and a footnote indicates that *p* < .01. To emphasize the significance of this difference, the authors state: "Nearly half of the probands had received psychiatric treatment as compared to one quarter of the controls" (pp.239-240); but for this item, actually $\chi^2 = 3.0034$ with *p* = .083. The next line of the table offers an opposite example: eight probands and two controls received psychiatric hospitalization, for which a *p* < .05 is shown; but $\chi^2 = 5.0475$ and *p* = .025. In Table 4, for the items hallucinations, lost control, and morning drinking, 3, 19, and 16 probands and 0, 13, and 9 controls exhibited the respective symptoms with *p* values of < .05, < .02, and < .02; but $\chi^2 = 2.2303$, 4.7069, and 5.4114 with *p* values actually equal to .135, .030, and .020 respectively. Further, in the text itself, the authors note that 16 of the proband parents and 10 of the control parents had been arrested at some time

for drunkenness, with $\chi^2 = 4.441$ and $p < .02$; but the value of p is .035. That is somewhat weaker support for "the validity of the hospital diagnosis of alcoholism in the parents of the probands" (p. 241).

Table 4 also shows that, at some time in their lives, 10 of the probands and four of the controls could be designated as alcoholics ($p < .02$); but $\chi^2 = 4.5320$, with $p = .033$. If the definitions of problem drinker and alcoholic are considered to overlap and these categories are combined, then there are 15 probands and 15 controls who manifested the appropriate symptomatology, to yield $\chi^2 = 0.7782$ ($p = .378$). At a minimum, one must conclude that a lesser significance should be attached to the previous p value of .033. This criticism has previously been made most cogently by Murray et al. (1983). They state:

If the cut-off point for abnormality is widened to include not just alcoholism but also problem drinking, then evidence for any genetic predisposition vanishes.

Indeed, the control adoptees were more frequently categorized as heavy or problem drinkers than the index adoptees (Table V). This finding contradicts the evidence of Kaj's twin study and Cloninger's adoption study (*vide infra*) that not only alcoholism but also milder alcoholic abuse is under some degree of genetic influence. Furthermore, this finding runs counter to all the evidence that heavy drinking and alcoholism are closely related. Could it be that Goodwin's findings are simply an artifact produced by the threshold for alcoholism accidentally dividing heavy drinkers in the index and control groups unevenly? (p.42)

In light of the present examination of this study, this interpretation seems likely to be more than the mere possibility suggested by Murray et al. (1983).

Goodwin et al. determined the current status of the adoptees with regard to the designation alcoholic; whereas in Table 4 the listing depended on whether the adoptee had met the criteria for at least a year at some time, they also assessed his current status and found that eight of the probands and three of the controls exhibited the appropriate alcoholic symptoms at the time of interview. This comparison they calculated as $\chi^2 = 4.8671$ (with $p < .05$), but the correct value is $\chi^2 = 3.5589$, with $p = .059$. How did they obtain the value of 4.8671? In this instance, the authors did not use the Yates correction, though their methods section asserts that they did.

Although it may be churlish to contest their arithmetic, the errors are not few in number and establish the likelihood that other errors, not visible in the data presented, also occurred. It must be concluded that any genetic disposition was indeed unproven.

The more general criticisms of this investigation, made by Cabaniss (1979) and by Murray et al. (1983), are concerned with the composition of the control group and with the diagnosis. Since 50 of the controls included in the study had no biological parents with a recorded psychiatric hospitalization, it would appear that they would have been sufficient in number, close to the 53 probands (67 minus 14). Yet these 50 were combined with a reported 28 (from 37) adoptees whose biological parents had been hospitalized for non-alcohol-related psychiatric illness. Murray et al. state: "One would have thought that the first group would have provided a 'pure' control, but for some unspecified reason, the two control groups were combined." They go on to ask: "Could this have been to increase the likelihood of finding significant differences? Since

certain psychiatric conditions associated with diminished alcohol consumption are also partly heritable, e.g., obsessional neurosis, this combined control group cannot be regarded as satisfactory" (p.41).

In a major study by Cloninger et al. (1981) employing a pool of 862 Swedish males adopted by nonrelatives before the age of 3, 151 of whom had some record of alcohol abuse (characterized as mild, moderate, and severe), the authors

distinguish two forms of alcoholism that have distinct genetic and environmental causes and differ in their association with criminality, severity of alcohol abuse, and the frequency of expression in biological mothers. Postnatal milieu determines the frequency and severity of expression of the common types of susceptibility in both men and women. In contrast, the less common type is highly heritable in men but is seldom expressed in mothers of affected men. (p.861)

The adoptees comprised an unselected population, one in which no specific types of parents or children were sought, and included all illegitimate children born in Stockholm between 1930 and 1949 and adopted. Because of the range of this population, the investigators studied "how adoptees with particular types of congenital background react to different types of adoptive-placement. This is called a cross-fostering analysis because each possible combination (cross) of genotype and environment is studied" (p.862).

Extensive data about the adoptees were obtained from child welfare, Temperance Board, criminal, and National Health Insurance registers, and from the records of psychiatric hospitals. A discriminant function analysis yielded background variables that set apart the alcohol abusers from all others. Various other statistical procedures were also used in a complex and sophisticated analytical scheme. The manner of data presentation makes it difficult to assess the extent to which errors in arithmetic or in statistical assessment occur, but in their initial paper (Cloninger et al., 1981), and in their following paper (Bohman et al., 1981), there are errors in the calculation of chi square (see especially p.966, e.g., significance of the fourfold increase in alcohol abusers among 29 daughters of mothers who abused alcohol compared to the daughters with neither biological parent an alcoholic). Although the authors conclude that their results "reconcile" earlier adoption studies (Roe; Goodwin et al.), this reconciliation fails to take into account the unacceptability of these studies. Moreover, since Roe found no genetic influence, Goodwin et al. found genetic influence for alcoholism but not for problem drinking, and Cloninger et al. found genetic influence for problem drinking but not for alcoholism, these contradictory results would appear to require a new definition of "reconciliation."

Cloninger et al. discerned no continuum of genetic influence. Given the categories of mild, moderate, and severe abuse, they concluded that "environmental factors largely determine severity of abuse given a genetic predisposition to either mild or severe abuse"; and that "most moderate alcohol abusers had another distinct kind of predisposition that was highly heritable in men regardless of their postnatal environment" (p.865). Their analysis also indicated "that unidentified factors or unspecified interactions were determining the development of severe abuse" (p.865).

The alleged implacability of alcoholism is highlighted by the extraordinary logic of the authors' discourse on the high prevalence of nonfamilial (sporadic) alcoholism: they hypothesize that if children's alcoholism is not reflected in any signs of vulnerability to alcoholism on the part of their biological parents, this is because the latter "are not themselves exposed by virtue of their drinking customs. Thus, *alcohol abuse itself is not a sensitive criterion of genetic susceptibility*" (p.867, emphasis mine). The authors thus posit a widespread genetic susceptibility to alcohol abuse that is not manifested because social custom inhibits contact with alcohol. In this manner, it is implicitly asserted that no weight should be given to the estimated 47% to 82% of alcoholics who had no alcoholic parents (Cotton, 1979). For an inherited trait not to occur in one's relatives seems most peculiar. What then is the weight to be given heredity? Here the authors cayil, saying:

the demonstration of the critical importance of sociocultural influences in most alcoholics suggest[s] that major changes in social attitudes about drinking styles can change dramatically the prevalence of alcohol abuse regardless of genetic predisposition. (p.867)

Unlike that of Goodwin et al. or that of Kaij, the Cloninger et al. study involved interviews with none of the adoptees, and relied exclusively upon official registers. As Kaij points out, reliance for the diagnosis of alcoholism on the data of the Temperance Board register is likely to introduce sample bias by identifying as alcoholics those individuals who are also psychopaths. Thus, Murray et al. are led to conclude, as one must, that despite the statistical armamentarium brought to bear on the data, "one has doubts about the validity of the original distinctions" (p.43).

5.4 GENETIC MARKERS AND ALCOHOLISM

About 20 years ago I suggested (Lester, 1966) that "an association of alcoholism with other characteristics known to be inherited would appear to offer some support for a biological factor in the etiology of alcoholism" (p.420). Among these inherited characteristics are the blood groups and other serum proteins, phenylthiocarbamide taste sensitivity, defects of color vision, and (non)secretion of ABH blood group substances into the saliva. The "association" essentially says that since the genetic marker is inherited, so too, in some fashion, is the disease. People afflicted with cancer of the breast, the colon, or the rectum or with substantial hypertension do not appear to possess differing blood group frequencies from people without these diseases. However, people with duodenal ulcers have a higher frequency of blood group O; the relative incidence of this disease is not different among those in A, B, and AB blood groups, and it is estimated that the likelihood of getting duodenal ulcers is some 40% higher for blood group O than non-O individuals. (Stern, pp.624-627) Similarly, it is surmised that individuals who taste phenylthiocarbamide (PTC) as bitter are less likely to develop nodular goitres than nontasters of PTC.

Swinson (1983) has reviewed published reports, beginning in 1959, that examined the association between genetically determined characteristics and alcoholism. These genetic markers include the ones named above. Swinson concludes that there is no association between alcoholism and blood groups,

and that any significant association between alcoholism and the three other polymorphisms may well result from prior alcohol intake: an effect, not an antecedent, of alcohol use. Swinson is guarded on this issue, suggesting that further investigation in this area might fruitfully lead to greater clarity, especially if family studies and prospective assessments were to be made. I have no reason to disagree with this assessment.

These three reviews (Cabaniss, Murray et al., and Swinson) represent excellent, even-handed, cautious, and careful probing of the evidence, and they suggest that genetic involvement in the etiology of alcoholism, however structured, is weak at best. My present examination of part of the evidence extends and, I believe, strengthens these previous judgments.

5.5 PHARMACOGENETICS AND ALCOHOL METABOLISM

Much attention has been paid to apparent pharmacogenetic support for the weight to be assigned heredity in the etiology of alcoholism. Many authors (e.g., Erwin & McClearn, 1981; Goodwin, 1982; Rutstein & Veech, 1978; Schuckit, 1984; Shields, 1977) have accepted the work of Vesell et al. (1971), which reported virtually absolute genetic control of alcohol metabolism. In two subsequent papers by Vesell (1972a,b), the original 1971 data on seven MZ and seven DZ pairs of twins were repeated, although, for reasons not made known to the reader, the Federation Proceedings paper (1972b) roughly doubled the rates of ethanol metabolism (mg/mL/hr) shown in the tables of the other papers! Other values (ID, age, and body weight) were unchanged. The conclusions reached in these papers remain unchanged (Vesell & Penno, 1983).

I apologize for the detailed textual analysis that follows; it is done with the hope that the reader will appreciate how important it is to scrutinize what so many others have accepted unquestioningly. I would hope that other chapters in this volume provide a similar intensive examination of their area.

Whereas the original paper and that in the *Annals of the New York Academy of Sciences* (1972a) reported a heritability of 0.98, the Federation Proceedings paper reported the heritability to be 0.99, albeit on the basis of different rates (in most cases doubling those previously reported) of ethanol metabolism. (Heritability was calculated as the difference between the variance of DZ and MZ twins divided by the variance of the former.) My own calculations of the heritability yield values of 0.98 and 0.973, respectively (employing the authors' definitions of variance and heritability). Unfortunately, the values for DZ twins are unacceptable in at least two cases. For example, as judged by the slope of the lines representing the disappearance of alcohol from the plasma (mg/mL/hr), twin F.D. metabolizes ethanol at more than twice the rate of twin P.D. (0.24 vs. 0.11), and twin H.H. metabolizes ethanol some 43% more rapidly than P.M. (0.20 vs. 0.14). Yet if the alcohol concentrations are extrapolated to zero concentration, twin P.D. manages to eliminate the same ethanol dose in less time than twin F.D. (4.5 vs. 5.0 hours), and twin P.M. eliminates the ethanol dose in 5.2 vs. 6.2 hours for twin H.H. The seeming paradox apparently arises from differences in absorption and distribution of alcohol, factors not taken into account by these investigators: although

these particular sets of twins had identical body weights, their heights may have differed, and thus so may their body water space. Whatever the reason, these values cannot be used. If they are excluded, the heritability decreases to 0.934, on the basis of the original data (for the data in Federation Proceedings, the value falls to 0.895). It would also appear reasonable to include only same-sex twins in the dizygotic group; using same-sex twins, however, leaves only three pairs of fraternal twins, and it would be ridiculous to perform further calculations with such data.

It is of some interest that, on the data of Vesell et al. (1971), heritability values for body weight can also be calculated — on the assumption, of course, that there is less difficulty weighing a subject than administering alcohol and following its temporal course. Since such a calculation must, perforce, exclude twins of opposite sex, only seven MZ twin pairs can be compared with five DZ pairs: the heritability is then 0.809 (for body weight). Unfortunately, a DZ female pair differs by 35 kg in weight and should be regarded as an outlier: if this pair is excluded, the variance of body weight is greater in identical than in fraternal twins!

In addition to these indicated difficulties with the Vesell et al. (1971) data, Wagner (1973) pointed out that these investigators did not consider intra-subject variation — that is, the repeatability of a particular subject's data from time to time — when estimating heritability. Wagner's calculations, using their data, provide a value of 28.0% for the intra-subject coefficient of variation, in agreement with similar data of Wagner and Patel (1972), but *greater* than the coefficient of variation among subjects. Wagner concludes, diplomatically, that "This does not appear to be theoretically sound for a large population, but for the small amount of data available one obtains that answer" (p.173).

There are two major problems with the cited experiment. The first problem, which affects other similar investigations, is the confounding effect of differences in body water and in absorption of alcohol from the stomach. To obtain reliable results for the rate of disappearance of alcohol, the solution is to administer alcohol intravenously. The second problem is that only same-sex fraternal twins should be used and that the number of twins should exceed seven by at least a magnitude (Martin et al., 1981).

These problems are also evident in the work of Luth (1939), though to a lesser degree. (Vesell et al., 1971, were apparently unaware of their predecessor of 32 years, a situation not yet rectified in Vesell & Penno, 1983.) Luth studied the rate of ethanol elimination in 10 pairs of identical and 10 pairs of same-sex fraternal twins. Although he concludes that there is a genetic component (by comparing the mean intra-pair differences between the groups), he did not calculate the heritability. Such a calculation yields values of 0.63 for ethanol elimination and 0.67 for body weight. With larger numbers of twins for the calculation of the heritability of body weight, a value of about 0.8 is ordinarily obtained (never 1.00).

In 1977, Kopun and Propping carried out a careful study of 19 identical and 21 fraternal twin pairs, also assessing intra-subject variation in ethanol metabolism. Their heritability value for ethanol elimination (mg/mL/hr) was

0.46, for absorption ($\text{mg/mL}/30 \text{ min}$) was 0.57, and for ethanol degradation (mg/kg/hr) was 0.41. They concluded that these parameters, "although to some extent under genetic control, [are] appreciably influenced by environmental factors" (p.344). They attribute the high values of Vesell et al. (1971) to the relatively small number of twin pairs and to their inhomogeneity with respect to sex and age. Kopun and Propping are sufficiently self-critical to realize the problems of their own study: thus they find greater concordance for the absorption of alcohol in their identical twin pairs than for the intra-subject measure in single subjects — "We can only speculate and cannot explain these findings" (p.343). When other time intervals (between 30 and 60 minutes) were used for estimation of absorption, "no influence of genetic factors could be established" (p.343). It seems essential in such studies to remove the confound of absorption by giving alcohol intravenously and, as Kopun and Propping imply they understand, to give the dose of ethanol based upon the volume of body water: that is, to take account of age and height differences in addition to those of weight (Watson et al., 1981). (The concentration of alcohol circulating in specific tissues is a function of their water content — e.g., blood is 80.7% water — and the concentration in the body water depends on the amount of absorbed alcohol and the total volume of body water. The body water in males is a function of age, height, and weight, independent of age in females [Watson et al.], and averaging about 58% in males and 47% in females.) The clear necessity for such experimental rigor is underlined by the fact that, in a later paper, reporting the greater concordance of the EEG in MZ versus DZ twins after alcohol ingestion, Propping (1978) remarks that "during the period of the main effect of alcohol, i.e., between 60 and 180 min after alcohol intake, blood alcohol curves of MZ twins are not more similar to one another than those of DZ twins" (p.94).

Martin and his co-workers (1981; see also Gibson & Oakeshott, 1981) have embarked on a broad-ranging study of a similar, though more encompassing, nature. By 1981, they had already tested 79 pairs of 18- to 35-year-old twins and aimed "to collect data on at least 200 pairs of twins" (p. 90). These pairs, fraternal and identical, attend an 8-hour test session during which they receive 0.75 g ethanol/kg about one hour after a light, nonfatty breakfast. A variety of measures are made, including the absorption and elimination of ethanol, psychomotor and cognitive tasks, and hematological and biochemical assays some of which have been suggested to be indicators of previous experience with alcoholic beverages. The authors make clear their view that previous studies in this area (Luth, 1939; Vesell et al., 1971; etc.) "involving fewer than 100 pairs have a very low probability of providing a critical test of relevant hypotheses when used for traits that are incompletely inherited" (p.90). Unfortunately, at least for the rate of alcohol elimination, their use of oral administration of alcohol may also confound the results they will obtain. In their 1981 paper, they offer a preliminary analysis of the effects of alcohol on an arithmetic computation task, concluding: "It is clear that if there are genetic differences affecting performance deterioration under alcohol on this task, then they are of small effect compared with individual environmental experiences and with the genetic differences that affect performance on all

occasions, regardless of whether alcohol is present or absent" (p.95). I can only join these authors in the hope that their analysis of data on the projected 200 twin pairs will provide valid and acceptable values on genetic involvement in some of alcohol's effects. For the present, however, the conclusion offered by Gibson and Oakeshott (1981), co-investigators of Martin, seems appropriate: "*In toto* the published studies on the inheritance of alcohol metabolism in man emphasize more the inadequacies of available data than provide insight into the causes of the variation" (p.131).

This conclusion was drawn despite the seeming acceptance by Gibson and Oakeshott of reports of higher levels of blood acetaldehyde after alcohol ingestion in siblings or sons of alcoholics compared to controls with no familial alcoholism (Schuckit & Rayes, 1979). Such higher levels of acetaldehyde have also been considered the basis of the "flushing" reaction, an inborn reaction to alcohol with presumably aversive consequences. The evidence that an aldehyde dehydrogenase deficiency is responsible for elevated levels of acetaldehyde and flushing in certain Orientals (Okada & Mizoi, 1982; Tsukamoto et al., 1982) and other ethnic groups is also on not particularly firm ground until methodological problems associated with the analysis for acetaldehyde are rectified. But that the conclusion of Gibson and Oakeshott remains warranted is made plain by the recent review of blood acetaldehyde levels by Eriksson (1983). Eliminating the sources of artifactual production of acetaldehyde has produced a situation wherein "acetaldehyde is not, at least with current analytical procedures, detectable in human venous blood during ethanol intoxication in subjects having neither a deficiency in, nor inhibition of, aldehyde dehydrogenase activity" (pp.147-148), a conclusion buttressed recently by Nuutinen et al. (1984). Even in subjects who might have such a deficiency, Eriksson's Table 1 makes clear that the tenfold higher blood acetaldehyde levels in Japanese flushers compared to Japanese nonflushers and the fivefold higher levels in Japanese flushers compared to Japanese alcoholics is still subject to analytical caveats. The negative association between flushing and frequency of drinking has, however, been reported to be significant only for Japanese men, not for Japanese women (Schwitters et al., 1982); these investigators conclude that "once persons drink at all, whether flushing occurs following the use of alcohol has only a trivial effect on drinking behavior" (p.1262).

Much the same situation prevails with regard to ethnic differences (Caucasians vs. Inuit vs. other Native Americans, etc.) in rates of ethanol disappearance. Small numbers of subjects, inattention to state of alimentation, and the confound introduced by the oral administration of alcohol raise serious doubts about the import of these studies for the involvement of heredity in alcoholism. More than a little weight must be given to the excessive use of alcohol by individuals "intolerant" to alcohol (flushers) and to the overwhelming numbers of "tolerant" individuals (nonflushers) who do not become alcoholics.

6. PRACTICAL IMPLICATIONS

The factors that enhance risk, or offer protection from risk, from nature and nurture are the meaningful objectives of inquiry. For the greatest part, the search for a genetic influence in alcoholism has cast no more light on factors identifying the individual at risk for alcoholism than did an earlier, different, and abortive attempt by Williams et al. (Williams, 1947; Williams et al., 1949a,b); the fault, to some degree, with Williams's approach was to disregard, in the alcoholic, alcohol-induced perturbations.

The possible causes and mechanisms whereby alcoholism develops have been set out by Clare (1979), Goodwin (1976, 1979a,b,c), and Grove and Cadoret (1983), among numerous others. The list is long, encompassing many possibilities and some improbabilities (e.g., Sudduth, 1977) derived from biology, psychology, and sociology. From proposers entranced with heredity (Goodwin; Grove & Cadoret), the list is hardly short and its length implies that not one but a panoply of genes are necessary, especially if genetic theory is to embrace aspects of personality like shyness and social insecurity, à la sociobiology. I cannot but agree with the views of Richardson (1984), in an analogous context: "that the credence accorded the views is disproportionate with their theoretical and empirical warrant. It is, furthermore, suggested that the proper explanation for the attraction and persistence of such views lies in their conformity with ideological norms" (p.396). Richardson might well have added "propagation" to "attraction and persistence."

A revealing, important, and most recent example is the publication by the U.S. National Institute on Alcohol Abuse and Alcoholism (1985) of a 39-page pamphlet titled *Alcoholism: An Inherited Disease*. One of the authors of the preface to the pamphlet is C. Robert Cloninger; the preface is an unabashed paean to the importance, value, and social impact that knowledge of the genetic factors in alcohol abuse and alcoholism will have for prevention and treatment. The preface assumes, and the remainder of the pamphlet assures the reader in most uncritical fashion, that the evidence is "impressive" for the genetic transmission of alcoholism. The pamphlet stresses the importance of genetic factors in the "majority" of alcoholics, and follows the logic previously alluded to: the high prevalence of nonfamilial alcoholism (hardly "sporadic" at a frequency of between 47% to 82%) is because the social customs of parents disallowed contact with alcohol. This is a most curious inhibitor in societies in which the drinking of alcoholic beverages is engaged in not simply by a majority but by an overwhelming majority of the population. One example must suffice to illustrate the bias: to show how impressive is the evidence for a genetic factor, the pamphlet informs the reader that "[Goodwin's] study was based on a large sample population of 5,483 individuals who had been adopted in early childhood" (p.4)—a contrast to the 133 (maximum) adoptees who were actually studied. Needless to say, the review by Murray et al. is not mentioned, nor is the admonition given by Cloninger et al. (1981) that "the demonstration of the critical importance of sociocultural influences in most alcoholics suggest[s] that major changes in social attitudes about drinking styles can change dramatically the prevalence of alcohol abuse regardless of genetic

predisposition" (p.867).

The pamphlet provides a rationale for genetic theory and alcoholism: as stated in the preface, it is not only that "Knowing that alcoholism is heritable should make it easier for us to rethink our cultural attitudes toward alcoholism and to accept it for what it is," but that alcoholism is "a disease with a molecular basis" (p.v). Reducing alcohol abuse and alcoholism to a molecular level means that one need no longer tinker with social and cultural attitudes; rather our efforts will be rewarded by designing "specific pharmacological interventions to prevent it and treat it" (p.36). These expressions find a ready acceptance by most alcoholics, and such notions at least dilute the responsibilities of the individual for the condition and diminish felt guilt.

Unrelated human beings, differing in many genes but holding untold numbers of genes in common, display similar onsets, courses, and outcomes in relation to alcohol; it is not unreasonable to believe that alcoholism is the result of those human beings' biology *and* the personal and social circumstances of their existence, external to but yet created by them. How then can the fate be fate, let alone implacable, if only a small portion of identical twin pairs share an alcoholic destiny? Despite the identity of 100% of the genes in each member of such a pair, and a far higher common nurture than fraternal twins, the fact of the existence of a nonalcoholic co-twin argues for more than the effect of one or more heritable traits. Nurture, in the larger sense of its intertwining with the genotype, must play a far greater and more decisive role than the proponents of heredity appear willing to concede. Furthermore, even if it were possible to partition such contributions without error in particular populations, we would still not know how to act absent an etiological mechanism.

Scientific inquiry would be the victim if any simplistic ideology of genetic theory and alcohol abuse and alcoholism were not questioned. And the spirit of the scientific enterprise would be diminished if this, or any other alternative perspective, were not criticized.

I believe that no elucidation of the etiology of alcoholism is possible without a prospective longitudinal study embracing the assessment of probable mechanisms from the biological to the sociocultural. Such a holistic approach rejects the simplistic and reductionist search for a single cause, the "Thomas switch" that Goodwin (1982) admires and apparently expects will one day appear, ready to turn alcoholism, like syphilis, "on and off" (Thomas, 1979). To believe this is truly to believe in the implacable fate. It is noteworthy that the examples given by Thomas are tertiary syphilis, chronic tuberculosis, and pernicious anemia, the switches being the spirochete, the tubercle bacillus, and a vitamin deficiency, all causes *external* to the organism. Unless we are asked to believe that alcoholism arises from an analogous external cause, the comparison may be inapplicable. Or is the availability of alcoholic beverages the cause, and prohibition the switch? If something external to the organism can be modulated in some manner, say by decreasing the effect of alcohol, will this eliminate alcoholism?

It does not appear that Goodwin would accept such an external mechanism. For genetics to have a place in such a schema, something internal to the organism must be switched. Since Thomas believes also "that schizophrenia

will turn out to be a neurochemical disorder, with some central, single chemical event gone wrong" (p.169), I assume that Goodwin too expects the rectification of a neurochemical event to transform the alcoholic; both would appear to accept the heritability of schizophrenia despite the steady decrease in the estimate of its value over the years. In 1980, Cassou et al., after an impressively lengthy and exhaustive reevaluation, concluded that there was a total absence of proof of a genetic effect in the schizophrenic process. Then, in 1981 and again in 1983, Lidz and his co-workers came to the same conclusion, that "the investigators' more direct study of the adopted-away offspring of schizophrenic parents [failed] to find statistically significant evidence of a genetic influence" (1983, p.434). Given the interactions and interrelationships of the increasing numbers of presumed neurotransmitters, Thomas's exuberance appears overdone; the nature of the genetically related neurochemical has, so far, been sought in vain.

7. RELATIONS TO OTHER THEORIES

It is my belief that a theory involving the interaction of genetics and environmental influences is compatible with any of the theories elaborated in this volume. I thus assert that no single factor or event "causes" alcoholism — that it is, to use what is to some an overworked phrase, a disease, syndrome, or process of a multifactorial nature. And its etiology will be found, variously, in the results of investigations from the areas with which this volume deals, from neurobiology and neuropsychology to politics and economics. I also believe that only the kind of longitudinal prospective study previously described, asking the right questions, will provide the answers.

8. CONCLUDING COMMENTS

Scientists in this area commonly focus on the relatively small number of genes that confer biological individuality (Williams, 1956), on those genes that divide us, for example, into the races of humankind. But our common genetic endowment, the result of a long biological history, would seem to be the great base upon which our culture and social history acts. There will then be many "switches," turned on and off, in the complex network of interactions between the individual's biology and culture that produce the alcoholic process.

The alcoholic is not doomed by any immutable laws of nature. The individual at risk for alcoholism has available many compassionate helping hands provided by society. However described, whether disease, process, or syndrome, whether physical and/or mental, the individual can also stand with Henley (1875/1944) and say:

It matters not how strait the gate,
How charged with punishments the scroll,
I am the master of my fate;
I am the captain of my soul.

understanding that few captains or masters ever operated without a helping crew to keep the ship from foundering.

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2. A NEUROBIOLOGICAL THEORY OF ALCOHOLISM

Boris Tabakoff and Paula L. Hoffman

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1. INTRODUCTION

The hope of every researcher has been that parsimony governs the laws of nature and of human behavior. This hope may prove to be a reality in the realm of laws of nature, but human behavior, particularly with respect to the use and abuse of alcohol, may not conform to parsimonious explanations. Thus, when concerned with the presentation of a theory to explain "alcoholism" at any level, we are confronted with a number of caveats that tend to limit significantly our ability to present a universally satisfactory hypothesis.

The neurobiological hypothesis presented in this chapter addresses only a segment of the multitude of phenomena that both define and result from "alcoholism." Since the whole area of neurobiology, as related to alcoholism, is still in its infancy, most of the available data are derived from studies with animals and one can only extrapolate from this work to try to describe the human condition.

Many definitions of alcoholism (Jellinek, 1960; WHO, 1952) include the terms *reinforcement*, *tolerance*, and *physical dependence*, and also concern themselves with the organ pathologies that result from excessive use of alcohol. Positive reinforcement has been considered as the major reason for maintaining drug-taking behavior, and tolerance and physical dependence can be clearly demonstrated in both animals and humans as manifestations of chronic

ingestion of high doses of ethanol. Reinforcement, tolerance, and physical dependence will also constitute the primary phenomena that we will use to develop the neurobiological hypothesis of alcoholism.

A positively reinforcing stimulus is one that, when its presentation is contingent on a specific response, leads to an increased rate of generation of that response. One could, therefore, contend that if the ingestion of ethanol were reinforcing to an individual, that individual would perform the necessary behaviors to provide him or herself with ethanol.

Tolerance can be defined, in the most general terms, as a diminished effect of a particular dose of ethanol after prior exposure of an organism to ethanol. A companion definition of tolerance is that a greater dose is necessary to achieve a certain level of effect in an individual after prior ingestion of ethanol, as compared to the dose necessary to achieve the effect on initial exposure. Terms such as "innate tolerance" or "initial tolerance" have been used, in some cases, to describe an individual's sensitivity to an initial dose. However, we will reserve the term "tolerance" for *changes* in sensitivity as a result of ethanol ingestion, and will use the term "initial sensitivity" to describe an individual's sensitivity to the first dose. One can be more precise and consider initial sensitivity as the individual's response to the early phases of first exposure — for example, during the rising phase of the blood ethanol curve. Tolerance (acute tolerance) has been shown to develop even during the time that a single dose of ethanol is being metabolized by the body.

Physical dependence has been defined primarily by the presence of a characteristic set of symptoms that appear when the intake of ethanol is abruptly terminated. These symptoms are, in many cases, opposite to the signs of acute intoxication. It should be stressed that the definition of physical dependence is based on phenomena appearing as a result of terminating ethanol intake and not on physiological signs present during intoxication. Both tolerance and physical dependence can be considered as attempts of the CNS neurons and possibly glia to adapt to, or counteract, the disturbance in their external and internal milieu produced by ethanol.

An individual's initial sensitivity to ethanol and the development of tolerance will be viewed in this chapter as etiological factors contributing to the generation of pathological drinking behavior. Physical dependence will be viewed as one of the negative consequences of the pathological consumption of ethanol. Our approach will be to present the behavioral and physiological experiments that indicate the involvement of a particular neurochemical system in mediating or generating ethanol's actions. We will then examine the reported effects of ethanol on neurochemistry to ascertain whether these reports add credence to a contention that certain neurochemical phenomena are of importance in the actions of ethanol or in the quantitative control of ethanol intake.

It is assumed that ethanol is ingested for its pharmacological effects, and that certain of these effects are reinforcing and will maintain consumption. Although important caveats regarding this assumption will be detailed below, the reinforcing effects of ethanol have been demonstrated in a number of studies with animals, and the self-administration of ethanol can be manipu-

lated by procedures shown to be efficacious in manipulating the self-administration of other reinforcing drugs (Winger et al., 1983). There are, however, significant quantitative and qualitative differences between the reinforcing properties of ethanol and of other drugs such as opiates or amphetamines. Winger et al. (1983) indicate that the major differences between ethanol and other abused drugs, in terms of self-administration experiments, can be explained by *ethanol's lack of potency* relative to other drugs of abuse. Whereas most abused substances are administered at doses of $\mu\text{g}/\text{kg}$ or mg/kg body weight, ethanol has to be ingested or administered within the dose range of g/kg to obtain evidence of reinforcing effects or other effects related to an animal's behavior. At these doses, the aversive or tissue-irritating effects of ethanol, or its metabolites (e.g., acetaldehyde), may intervene to counter its positive reinforcing properties. The *balance* between an animal's or human's initial sensitivity to its positively reinforcing effects versus its aversive properties may be of primary importance in determining the result of initial experiences with ethanol.

The biochemical locus determining initial sensitivity to ethanol in the central nervous system (CNS) can be envisioned to be the milieu of the neuronal membrane. Since ethanol is an amphipathic substance (with affinities for both lipid and water environments), it would be expected to enter the lipid environment of cell membranes and to disrupt the normal order of the bulk lipids or, more important, to disrupt the normal interactions between the membrane lipids and the functional proteins (i.e., receptors, enzymes, ionophores) that reside in the neuronal or other cell membrane. The perturbation of the milieu of the membrane-bound proteins would, in turn, be expected to alter neuronal function and produce the physiological concomitants of ethanol-induced reinforcement, intoxication, etc. Several neuronal systems—that is, those using norepinephrine (NE), dopamine (DA), serotonin (5-HT), and enkephalin (ENK) as neurotransmitter substances—have been considered to be important in mediating the reinforcing effects of ethanol, and we will review the effects on ethanol on certain of these systems.

An alternative to the direct actions of ethanol itself on the elements of the CNS is contained in the hypothesis that the metabolism of ethanol in the body generates biologically active entities—acetaldehyde, tetrahydroisoquinoline (TIQ), and beta-carboline alkaloids—that, in turn, act at neuronal sites to produce reinforcement or certain of the other manifestations of ethanol ingestion.

Regardless of whether ethanol or ethanol-derived metabolites are the initial perturbing agents, we will assert that the CNS will alter its structure or function to resist certain of the perturbing influences of ethanol ingestion. This acquired resistance would manifest itself as tolerance to ethanol. Although the most current biological explanation for the development of tolerance is related to an adaptive change in neuronal membrane lipids (Chin & Goldstein, 1977), this explanation does not well account for the important role that learning or conditioning has been shown to play in the development of tolerance. We have, in the past, categorized tolerance into several interacting components (Tabakoff et al., 1982), and we will use this categorization in our further

discussion of tolerance. Tolerance can be considered as a positive feedback loop with regard to a person's *intake* of ethanol. Although tolerance would per se diminish certain effects of ethanol, it could promote greater intake to overcome the diminished effect. Tolerance development could also remove the aversive consequences of intoxication and, in this manner, *allow* for increased consumption.

2. THE HYPOTHESIS

In presenting the hypothesis, we will consider factors that determine the initial response to ethanol and we will emphasize that neuroadaptive processes play an important role in the etiology of "alcoholism." The motivation for a drinker to consume ethanol is initially controlled by expectation regarding the consequences of ingestion and a balance between that drinker's sensitivity to the reinforcing and aversive effects. In a drinker, the generators of cues for the expected effects can be envisioned to reside, in part, within *genetically determined* characteristics of the neurons of the CNS, particularly the DA, 5-HT, NE, and ENK neurons of the brain. Since ethanol does not have specific receptors and is expected to interact to an extent with all neurons of the brain, a mosaic of its actions has to be considered, rather than its action on one or another neuronal system.

As ethanol consumption is continued, and even during the time that the first dose is present in the body, the propensity of the individual to develop tolerance to certain of the effects allows for, or necessitates, the consumption of larger quantities to attain or maintain the desired pharmacological effect. The development of tolerance can be considered as an alteration in the pattern of the original mosaic of neuronal sensitivities. The altered neuronal sensitivities would shift the balance towards the greater intake. The ingestion of the larger quantities promotes the further development of tolerance and places the individual at risk for developing ethanol-related organ pathologies and physical dependence. In turn, the organ pathologies and physical dependence may become additional factors that modulate ethanol intake.

In summary, one can contend that, on the neurobiological level, three types of factors determine the consumption and the effects of ethanol during a person's drinking history. The first group of factors is concerned with generating the motivation to consume ethanol. The second group of factors is concerned with the neuroadaptive consequences of consumption, or, stated another way, the second group of factors is concerned with the ability of individuals to alter their physiology in response to ethanol. The third group of factors relates to whether the neuroadaptive consequences of consumption form positive feedback loops to promote excessive intake.

3. RELATION TO OTHER THEORIES

The major tenet for developing a neurobiologically based theory of alcoholism is that biological individuality, as related to initial sensitivity to

ethanol and the propensity to develop tolerance, is an important factor in the etiology of alcoholism. Studies of family history of alcoholism (Cotton, 1979), concordance for alcoholism between twins (Kaj, 1960), and more recent studies of the influence of adoption and environment on development of alcoholism (Cloninger et al., 1981; Goodwin et al., 1973; Schuckit et al., 1972) all suggest clearly that development of alcoholism can, in many cases, be related to genetically transmitted predisposing factors (but see discussion in Chapter 1). Since genes code for chemical entities that in turn constitute the structural and functional components of the body, one would assume that the individual predisposed to alcoholism possesses the constellation of genes and the characteristic chemistry that would promote the development of alcoholism once the individual begins to consume ethanol. The preceding statement should not be taken to indicate that only a single gene or even a specific set of genes predisposes the heterogeneous phenomenon we refer to as alcoholism. For instance, studies with mice indicate that individual sensitivity to even a "simple" behavioral effect of ethanol, sedation, is determined by the combined actions of nine to thirty gene loci (Dudek & Abbott, 1984).

If one accepts (and one should, on the basis of the ample evidence) the premise that the genetic constitution of an individual is an important factor in the development of alcoholism, then one naturally should ask what are the attributes that are being inherited and which of these attributes may be of greatest importance in the development of alcoholism in the population as a whole or, at least, in a significant subset of individuals. The neurobiological theory expressed herein considers that genetically determined sensitivity to the aversive and reinforcing effects of ethanol is a critical factor in the etiology of alcoholism. We, however, contend that the ability of ethanol to generate reinforcement is under the influence of another genetically determined factor. This factor is the propensity of the individual, and particularly the brain of that individual, to develop a resistance to the aversive or the *reinforcement-diminishing* properties of ethanol. Because of this developed resistance (tolerance), the positively reinforcing effects predominate.

The central role for reinforcement in the neurobiological theory of alcoholism allows for consideration of alcoholism in a context similar to that used to describe addictions to other chemicals. The addictive properties of opiates and of stimulants such as amphetamine and cocaine have been related to the reinforcing properties of these compounds (Schuster, 1978; Woods & Schuster, 1971). Although such a unifying hypothesis of addiction is appealing, the text of this chapter will indicate that the biological mechanisms by which ethanol produces reinforcement may differ from the proposed mechanisms (Goeders & Smith, 1983) by which opiates or cocaine produce reinforcement. The different mechanisms of action of the various drugs would not obviate the importance of the terminal physiological event (i.e., reinforcement), but involvement of different neuronal systems in mediating the reinforcing properties of ethanol, as compared to systems mediating reinforcing properties of other drugs, implies a specificity that would be important in considerations of cross-addictive potentials of drugs and prevention and treatment strategies.

The proposed involvement of neuroadaptation (i.e., development of tolerance) in the expression of ethanol's reinforcing properties necessitates the consideration of learning and memory as important contributors to the etiology of alcoholism. We have, in the past (Tabakoff et al., 1982) and in this chapter, considered developed tolerance to ethanol to be an example of a form of biological memory. In this sense, the massive literature on the biological substrates of learning and memory becomes applicable to the consideration of the proposed hypothesis. The major distinction between prior theories that have invoked learning to be a central event in the development of addictions and our theory is that our current hypothesis stresses the interaction of two distinct biological memory processes in the etiology of alcoholism. The first memory-related process would be the development of tolerance to certain aversive effects of ethanol, and the second memory-related event would be the encoding of information about its reinforcing properties. Prior theories relating memory processes to the addictive properties of drugs, particularly of the opiates (Siegel, 1979; Wikler, 1973) have primarily concentrated on the importance of associative processes (conditioning) in the expression of reinforcement or certain other characteristics of addiction (e.g., craving, withdrawal symptoms). In our subsequent discussion we will consider at length the role of conditioning in the development of ethanol tolerance.

The importance of conditioning in the development of ethanol tolerance emphasizes the fact that an individual's chemical (biological) constitution does not function in a vacuum. Environmental (exogenous) factors interact with endogenous chemistry in definitive ways. Thus, even the most abstract theories from a biologist's point of view (e.g., alcohol pricing structure and governmental controls of alcohol availability as etiologic factors in alcoholism) can be accommodated if one wishes to generate an all-encompassing theory. We do not have aspirations to develop such a theory, and thus, while we fully appreciate the importance of environmental factors, we will concentrate our discussion and hypothesis on the biological portion of the alcoholism process.

4. CHARACTERISTIC RESEARCH METHODS

The field of physiological psychology and the numerous disciplines that constitute the neurosciences have contributed to the methodology described in our chapter. The basic research approach applied to generating the results we use for supporting our hypothesis has been the attempted correlation between neurochemical and neurophysiological events and behavior. To generate information on the neurochemical effects of ethanol, most studies have been performed on non-human animals. In a portion of such studies, ethanol has been added to brain tissue *in vitro*, and the effects of ethanol on receptors, enzymes, and structural components (e.g., neuronal membranes) of cells have been catalogued. Extrapolation from such studies is obviously difficult, since at present one can only speculate about which neurochemical systems are responsible for the various behavioral effects of

ethanol. The integration of the various neuronal systems that is so important to the *in vivo* situation is also disrupted in the process of obtaining tissue for study. Such studies have, however, provided insights into the molecular mechanisms of ethanol's actions.

The alternative approach has been to administer ethanol to an animal, record the behavioral consequences, and then examine brain tissue for ethanol-induced perturbations of function. Substantial difficulties arise in assessing brain chemistry at the time a particular behavior is being exhibited, since in most studies the behavior is terminated by the process of obtaining the tissue for chemical assays. To overcome certain of these problems, investigators have attempted to assay the chemicals appearing in extracellular fluids of the brain (CSF) by means of indwelling cannulae, thus allowing for *in vivo*, albeit indirect, means of assessing the effects of ethanol on neuronal chemistry.

The effects of ethanol on particular brain structures and neuronal pathways have also been examined by use of electrophysiological techniques that assess the electrical activity of areas of brain, populations of neurons, or single cells. Certain of these electrophysiological studies have attempted to correlate the electrophysiological effects with behavioral sequelae, but many have been aimed primarily at describing differences in sensitivity of particular brain areas to ethanol's actions and at delineating the mechanism by which ethanol depresses neuronal function. As with studies that utilize neurochemical techniques, electrophysiological studies would be of greater value if defined systems known to mediate a particular behavior could be studied. Even more seminal would be the generation of an understanding of which systems are of importance in the etiology of alcoholism. If these systems could be identified, the generation of a neurobiological hypothesis of alcoholism would be vastly facilitated.

In attempts to clarify which of the neurochemically and neuroanatomically defined systems are participating in the expression of ethanol's behavioral actions, investigators have either physically destroyed or pharmacologically modified the function of particular neuronal systems. The premise of such studies is: if a system is integral to the mechanism by which ethanol produces an effect on behavior, the destruction or modification of that system would alter ethanol's effects. Such studies of the acute effects of ethanol have generated primarily controversy. However, this approach has yielded *some* consistent and provocative results with regard to its *chronic* effects, such as the development of tolerance. These studies will be described in detail below.

Another means of distinguishing the neuronal and neurochemical systems that may be important in mediating ethanol's actions is through the use of genetically inbred or selectively bred animals. Investigators using inbred animals generally attempt to correlate the extent of a behavioral effect of ethanol in a number of inbred lines of animals with the extent of effect on a particular electrophysiological or neurochemical parameter. An excellent positive correlation *could* indicate a causal relationship. Correlations between neurophysiological and neurochemical events and ethanol-induced behavior have also been attempted in lines of animals selected over a number of generations for a particular extreme behavioral response to ethanol. Such studies

presume that over many generations of appropriate selection (McClearn, 1967), the neurochemical/neurophysiological differences between lines would be those that are related to the differences in behavior. Such selection studies have been quite effective in characterizing the factors responsible for the sedative/hypnotic effects of ethanol.

A major deficiency in the literature regarding the neurobiological actions of ethanol is that most published studies have concentrated on describing the effects of high doses (hypnotic levels). As we will state several times below, extrapolation from such results to events occurring at sub-anesthetic doses is laced with uncertainty due to unknown characteristics of the ethanol dose-response relationships. Certain of the more recent studies of neurochemistry have, however, provided complete information on the ethanol dose-effect relationships. The character of neurobiological events that mediate particular behaviors that are evident at doses of ethanol well below those producing anesthesia have also recently been explored. We have relied heavily on these latter studies for the generation and support of our hypothesis.

5. HISTORICAL PERSPECTIVE

The neurobiological hypothesis has its origins in postulates that have been put forth to describe the physiological concomitants of acute and chronic administration and withdrawal of other dependence-producing substances (e.g., opiates, barbiturates), but is distinguished in that, in our formulation, attempts have been made to incorporate the fact that ethanol's actions are not initiated by its interactions with a specific set of receptors.

The observation of Olds and Milner (1954), that rats would learn arbitrary behaviors to obtain low-level electrical stimulation in some, but not all, areas of brain, led to the current view that the brain contains neuronal circuits that function to provide a rewarding consequence in response to certain stimuli. The reward pathways activated by electrical stimulation seem to be those that naturally provide reward for behaviors such as drinking, feeding, and sexual activity (Caggiula, 1970; Margules & Olds, 1962); and damage to particular brain areas (e.g., lateral hypothalamus) disrupts not only behaviors such as eating and drinking, but also the efficacy of electrical stimulation of that brain area to provide reward (Clavier & Routtenberg, 1976). From such studies derived the notion that, in addition to mediating the effects of natural rewards, such as food or sex, the brain reward pathways are important in mediating the rewarding effects of various stimuli, including drugs with high abuse potential. Esposito and Kornetsky (1978) have reviewed other information that has led to the hypothesis that drugs of abuse exert their reinforcing effects by acting on or through the neural pathways that respond to natural rewards.

The earlier studies on the reinforcing properties of the narcotic analgesics were extended to investigate the effects of ethanol on brain reward

systems. In general, such studies fell into two categories. The first category examined the effect of ethanol on the reinforcing properties of brain stimulation in animals. These studies have been directed at generating information as to whether ethanol, like other drugs of abuse, modulates the activity of systems important in providing reward for certain behaviors. The original studies in this category were performed by St. Laurent and Olds (1967) and led to the speculation that ethanol would facilitate electrical self-stimulation of certain brain reward sites, *but not others*. Since most subsequent studies (discussed below) have used the lateral hypothalamus as the site for placement of stimulating electrodes, this speculation remains untested.

In the second category of studies, operant conditioning techniques have been used to examine characteristics of ethanol self-administration by animals (Winger et al., 1983). In such studies, it is assumed that the drug in question has the ability, in and of itself, to activate the neuronal systems that are aroused by certain other rewards, and that the drug will be administered by the animal for its reinforcing properties, just as the animal would administer electric current to the neuronal systems mediating reward. A variant of the second category of studies is the use of a situation in which the animal is given a choice between a drug (e.g., ethanol solution) and one of the natural reinforcers (e.g., water) (Myers, 1972). The choice of the drug is taken to indicate that it possesses reinforcing properties. It is this latter type of study that has generated the most speculation regarding the neurochemical mechanisms important in mediating the reinforcing effects of ethanol.

Tolerance to and physical dependence on "addictive" substances have, in the past, been thought of as processes that had identical biologic determinants (Himmelsbach, 1943); but more recent studies have demonstrated that ethanol tolerance, and physical dependence on ethanol, can be independently modulated by neurotoxins (Tabakoff & Ritzmann, 1977) and neuropeptides (Hoffman et al., 1979). Early hypotheses used to describe ethanol tolerance and dependence were based on contentions that ethanol, like other drugs of abuse, would produce alterations in neurotransmitter metabolism or the receptors for neurotransmitters. These hypotheses resembled the "denervation supersensitivity" hypothesis of Jaffe and Sharpless (1968) and the "receptor proliferation" hypothesis presented by Collier (1966) to describe tolerance and dependence on barbiturates and opiates.

The original version of the hypothesis proposed by Jaffe and Sharpless (1968) was based on observations (Sharpless, 1964) that tissue (striated muscle) deprived of its innervation would attempt to counter the lack of stimulus by developing a greater sensitivity to the neurotransmitter (acetylcholine, in this case) that is usually released by neurons innervating the muscle. This adaptation would generate a rebound supersensitivity, expressed as exaggerated contractions, if the transmitter substance was, at a later time, applied directly to the denervated muscle. Jaffe and Sharpless (1968) conceptualized that sedatives such as the barbiturates would depress CNS neuronal function and thus, in essence, produce a temporary denervation. The cells usually receiving chemical stimulation from the depressed neurons would

attempt to adapt to the diminished input. The adaptation would, however, generate an inappropriate sensitivity if the CNS depressant was withdrawn and neuronal activity was disinhibited. The supersensitivity of certain of the elements of the CNS would generate the hyperexcitability and other symptoms witnessed during drug withdrawal. Although Jaffe and Sharpless did not specifically attribute the changes in sensitivity to alterations in cell surface receptors for neurotransmitter, Collier (1966) hypothesized that the hyperexcitability resulting from chronic perfusion of the gut with opiates was due to a proliferation of receptors in response to opiate-induced depression of activity. Current studies (Dum et al., 1979; Klee & Streaty, 1974) clearly demonstrate that opiate dependence is not characterized by an increased number of receptors for endogenous or exogenous agents having opiate-like biologic activities. However, the general principles elucidated by Jaffe and Sharpless (1968) and Collier (1966) have been the foundation of many neurochemical studies of alcohol tolerance and dependence. Adaptive changes in enzymes that mediate neurotransmitter metabolism have also been considered to participate in the development of tolerance to and dependence on ethanol, just as they have been considered as factors in the development of tolerance to and dependence on opiates (Goldstein & Goldstein, 1968).

The observation that ethanol may produce its effects, as do a number of other general anesthetics (Meyer & Gottlieb, 1926; Seeman, 1972), by intercalating into, and disrupting the order of, neuronal membranes led Hill and Bangham (1975) to propose that ethanol tolerance and physical dependence may be a result of adaptive changes in the physical characteristics of neuronal membranes that would produce a resistance in the system to the disruptive effects of ethanol. A number of investigators, notably Goldstein (Chin & Goldstein, 1977; Goldstein et al., 1982; Lyon et al., 1981; Perlman & Goldstein, 1984), have further developed the "membrane hypothesis" into the neurochemical explanation of the acute intoxicating effects of ethanol, and of the development of tolerance and physical dependence; this work will be detailed below. In these hypotheses, the direct effects of ethanol on neuronal membranes are thought to produce the stimulus for adaptive changes that result in tolerance and dependence (homeoviscous adaptation; Sinensky, 1974). Other studies, however, indicated that the presence of ethanol in the milieu of a neuronal system was a necessary but not sufficient event for the development of tolerance. Appropriate neuronal activity in the presence of ethanol seemed to play a significant role in "priming" the development of tolerance (Traynor et al., 1980). LeBlanc and co-workers (1973) concluded from their studies of tolerance development that changes responsible for tolerance to ethanol are influenced by the functional demand imposed on the CNS during the period of the drug effect. More recent studies (Crowell et al., 1981; Wenger et al., 1981) on tolerance development make a further case for the importance of environmental stimuli in the development of tolerance, and for the contention that tolerance to the behavioral effects of ethanol cannot be demonstrated without prior exposure of an animal to the effects of ethanol within the test

situation (i.e., tolerance is largely a learned phenomenon). Consideration of the full range of available experimental data (see below), however, indicates that both direct effects of ethanol on the neurons of brain and environmental contingencies play a role in the development of tolerance.

What has not been clearly conceptualized in the past is whether the development of tolerance to or physical dependence on ethanol would (1) actually produce changes in the function of neuronal systems that mediate *reinforcement*, or (2) affect the reinforcing properties of ethanol in an indirect fashion (e.g., by generating or diminishing an aversive stimulus that accompanies its intake). Two recent studies (Numan, 1981; Reid et al., 1985) on the importance of prior experiences with ethanol in the expression of its reinforcing properties have shed some light on this issue and aided us in the presentation of the relationship between tolerance and reinforcement.

In the above-mentioned studies regarding ethanol intake, reinforcement, development of tolerance, and development of physical dependence, ethanol was considered to be the factor responsible for generating the reinforcement and/or the tolerant/dependent state. However, an alternative proposal, in which a metabolic product of ethanol figures prominently in the development of a "preference" for and dependence on ethanol, has maintained some popularity.

Ingested ethanol is metabolized to acetaldehyde, and Cohen and Collins (1970) have shown that acetaldehyde can condense with catecholamine neurotransmitters to produce the tetrahydroisoquinoline (TIQ) alkaloids (e.g., salsolinol). At about the same time, Davis and Walsh (1970) demonstrated that incubation of homogenates of brain tissue with acetaldehyde resulted in the formation of even more complex TIQ alkaloids, which were derived from the condensation of the aldehydes produced by the oxidative deamination of the catecholamines and the parent amines. The initial condensation product that was isolated by Davis and Walsh (1970) was tetrahydropapaveroline (THP), and this alkaloid was hypothesized to be generated because of the competitive inhibition, by acetaldehyde, of the metabolism of the neurotransmitter-derived aldehydes in brain. Since THP is a precursor in the synthesis of opiate alkaloids by the opium poppy, Davis and Walsh (1970) hypothesized that a similar conversion of THP to dependence-producing opiate substances may take place in animals. Although significant controversy surrounds the TIQ hypothesis of alcohol dependence (Goldstein & Judson, 1971; Halushka & Hoffmann, 1970), an interesting current observation has kept alive the consideration of TIQs within the context of "alcoholism"-related phenomena. Myers and Melchior (1977b) reported that infusion of THP into the cerebral ventricles produced a significant increase in animals' free-choice consumption of ethanol. These studies will be elaborated upon in subsequent sections of this chapter. The work related to the effects of the TIQ alkaloids and other compounds, such as the beta-carbolines (Myers & Melchior, 1977), on ethanol preference remains a major area for speculation regarding the factors responsible for ethanol ingestion.

6. REVIEW OF RESEARCH METHODS AND RESULTS

6.1 EXPERIMENTS DIRECTED AT DETERMINING FACTORS RELATED TO INITIAL PREFERENCE FOR ETHANOL

6.1.1 *Studies on the Reinforcing Properties of Ethanol*

Several experimental paradigms, including intravenous, intragastric, and oral routes of ethanol self-administration, have been used in attempts to generate data on the reinforcing properties of ethanol. Its reinforcing properties have not been easily demonstrated in studies in which minimal prior exposure to ethanol occurs within the testing paradigm (Asin et al., 1985; Numan et al., 1984; van der Kooy et al., 1983). These observations contrast with data generated with other substances commonly abused by humans such as cocaine and opiates (van der Kooy et al., 1982), even though the experimental methods used to assess the reinforcing properties of ethanol (see below) have been similar to those used to demonstrate the reinforcing effects of many addictive chemicals. For instance, the use of conditioned place preference methodology, with which the reinforcing or punishing (aversive) effects of a number of agents have been demonstrated (van der Kooy et al., 1982), indicated no reinforcing effects in rats given ethanol doses ranging from 0.055 to 1.1 g/kg via the intravenous route, and doses of 0.5 to 5 g/kg via the intragastric route (Asin et al., 1985; van der Kooy et al., 1983). Only conditioned place aversion could be demonstrated, and this effect occurred with the higher doses used in these studies. The value of examining conditioned place preference as a means of discerning the initial reinforcing effects of a drug lies with the fact that subjects need little prior drug experience before one assesses reinforcement. The association between a drug effect and the distinctive environment of one arm of a maze can be established with one to four training doses of the drug. The authors of one of the above-mentioned studies (van der Kooy et al., 1983) speculated that prior training with ethanol, and development of tolerance to certain aversive effects of ethanol, might be necessary to unmask its positively reinforcing effects. Support for this contention can be generated by the recent studies of Reid et al. (1985), in which a prior period of chronic ethanol administration or ingestion allowed a conditioned place preference for ethanol to be evidenced.

Methodology in which an animal is trained to self-administer ethanol by intravenous (Winger & Woods, 1973) or intragastric (Altshuler, 1976) techniques has also indicated that ethanol has many of the reinforcing properties associated with other drugs commonly abused by humans (Winger & Woods, 1973; Winger et al., 1983). One should, however, consider that the period of training necessary to establish intravenous or intragastric self-administration of ethanol may be sufficient to produce tolerance to certain aversive consequences and allow for the reinforcing properties to be evidenced (Numan, 1981). Another suggestion presented by van der Kooy et al. (1983) was that ethanol may be viewed as a secondary reinforcer, and only when it is paired with effective primary reinforcers—food, sex, etc.—would its reinforcing effects be evidenced. Support for the second contention was presented in a

study by Stewart and Grupp (1981), in which rats demonstrated no place preference or aversion when given a 500 mg/kg (i.p.) injection of ethanol within the context of a conditioned place preference paradigm. When the same dose was administered in the presence of food, the rats demonstrated a strong place preference for the environment in which it was administered, compared to an environment in which an injection of saline was given with the food.

Further insight can be gained into the reinforcing effects of ethanol by examining its effects on systems mediating brain stimulation reward. Early reports indicated that ethanol would facilitate electrical self-stimulation at certain anterior hypothalamic sites (St. Laurent & Olds, 1967). More recent studies (Carlson & Lydic, 1976; Lorens & Sainati, 1978; Magnuson & Reid, 1977) have examined the effects of ethanol on rates of stimulation of the lateral hypothalamus, and have noted a stimulatory effect of low doses (about 0.5 g/kg in rats) on the rate of bar pressing for self-stimulation. The extent of the effects of this dose varied from a 25% increase (Magnuson & Reid, 1977) to an approximate 170% increase (Lorens & Sainati, 1978), without a change in the electrical threshold for self-stimulation (Carlson & Lydic, 1976). The inability of low doses to alter the electric current threshold for self-stimulation indicated that ethanol was not producing its effects on rates of responding by directly altering the function of neurons mediating the rewarding effects of the electrical stimulus. Carlson and Lydic suggested that the low doses of ethanol accelerated operant responding by activating a motor-facilitating or general arousal system of brain.

However, larger doses of ethanol (i.e., about 1 or more g/kg in rats) have been demonstrated to produce disruption of lever pressing behavior in brain self-stimulation paradigms (Carlson & Lydic, 1976; Magnuson & Reid, 1977). Doses in the range of 0.9–1.2 g/kg significantly raised thresholds for self-stimulation current, suggesting a suppressive effect of these doses on the neuronal systems that mediate the rewarding effects of self-stimulation (Carlson & Lydic, 1976). There is evidence that tolerance can develop to these high-dose direct effects of ethanol on brain reward systems. For example, when ethanol was administered to rats in a paradigm (Majchrowicz, 1975) that resulted in the development of physical dependence, and presumably tolerance, the effects of higher doses (1 g/kg) on rates of operant responding for brain stimulation were diminished, whereas the effects of the lower doses were unchanged (Magnuson & Reid, 1977). Ethanol could thus be considered in a context similar to other abused drugs. Studies of the chronic administration of cocaine or opiates indicate that animals are more likely to develop tolerance to drug effects that occasion a loss of reinforcement, rather than those effects considered to be positively reinforcing (Schuster, 1978).

6.1.1a Identification of neuronal systems that may mediate the reinforcing effects of ethanol. In considering the neuronal systems responsible for mediating the reward properties of electrical stimulation, attention has focused on the catecholamine (CA) neurons of brain. Wise and Routtenberg (1983) have reviewed the evidence that indicates the primary importance of dopaminergic neurons, whereas earlier studies had stressed the importance of

noradrenergic neurons (for a review, see Fibiger, 1978). The issue of which CA system is preeminent in mediating electrical- or drug-induced reinforcement is not yet fully resolved. *No studies* have directly assessed how either dopaminergic or noradrenergic neurons may mediate the effects of ethanol on brain-stimulation reward responding. Nevertheless, the suggestion that the rewarding properties of brain stimulation are mediated by brain endorphin systems (Belluzzi & Stein, 1977) led Lorens and Sainati (1978) to examine the effects of an opiate receptor antagonist, naloxone, on ethanol-induced changes in responding for brain stimulation. Their studies demonstrated that naloxone (5 mg/kg) could eliminate the facilitatory effect of ethanol on hypothalamic self-stimulation. Another experimental approach has also produced data regarding the possible importance of endogenous opiate systems in mediating the reinforcing effects of ethanol. In this approach, intravenous self-administration of ethanol was monitored in monkeys (Altshuler et al., 1980). Animals were selected for the experiment on the criteria that they had initiated ethanol self-administration spontaneously and were stable in their ethanol infusion behavior. The administration of naltrexone (1–5 mg/kg) 30 minutes prior to the daily access to ethanol resulted in a significant diminution of ethanol self-administration over the 15-day period of the experiment. The observed pattern of increased responding for ethanol followed by diminution of ethanol intake in animals treated with naltrexone resembled behavior typically seen during extinction of a positively reinforcing operant task. The investigators concluded that naltrexone was interfering with the reinforcing properties of ethanol, and was doing so by virtue of its opiate receptor blocking properties (Altshuler et al., 1980).

The greatest volume of studies, aimed at providing information on the neuronal systems that may mediate the reinforcing effects of ethanol, have been performed using paradigms in which the animal can choose to consume ethanol-containing solutions or water, but the use of such studies to draw conclusions on the reinforcing properties of ethanol has been questioned (Cicero, 1980). The major criticism pertains to whether rats, or other rodents used for such experiments, imbibe enough ethanol to produce any direct effect on the CNS. This criticism could not be answered until one knew the blood and brain concentrations of ethanol present in an animal consuming ethanol in a free-choice paradigm, and until one knew whether the consumed doses could alter CNS physiology. Recent studies indicate that ethanol directly affects the electrical properties of locus coeruleus (NE) neurons at levels as low as 5–10 mM (i.e., 25–50 mg%) (Shefner & Tabakoff, 1985), and such levels are easily found in the circulation of selectively bred, ethanol-preferring rats offered a choice between ethanol and water (Li & Lumeng, 1977). The demonstration that rats bred for a preference for a 10% ethanol solution will voluntarily lever press for an oral dose (Penn et al., 1978) provides further evidence that ethanol may be selected in a free-choice situation because of its reinforcing properties. Thus, studies that use self-selection of ethanol in attempts to determine systems responsible for its reinforcing effects should be given ample consideration.

Administration of dopamine-beta-hydroxylase inhibitors to rats was

shown to attenuate oral and intragastric ethanol self-administration (Amit et al., 1977; Davis et al., 1978). The effect of these agents was postulated to result from their ability to lower brain norepinephrine levels. However, a number of these agents (e.g., disulfiram, FLA-63) are also inhibitors of aldehyde dehydrogenase (Amit & Sutherland, 1975/6), and adverse consequences of increased circulating levels of acetaldehyde produced by aldehyde dehydrogenase inhibition can certainly reduce ethanol intake (Schlesinger et al., 1966).

In principle, the use of 6-hydroxydopamine (6-OHDA) to destroy noradrenergic neurons of brain could overcome the inadequacies attendant on the use of dopamine-beta-hydroxylase inhibitors; however, the results from this procedure have been equivocal. Certain studies utilizing 6-OHDA have demonstrated a diminished intake when ethanol solutions are offered as a choice against water (Amit & Sutherland, 1975/6; Mason et al., 1979), but others have demonstrated an increased ingestion of ethanol after administration of 6-OHDA (Kianmaa et al., 1975). Major differences in experimental design can, however, be noted between studies. Destruction of specific noradrenergic projections (Kianmaa et al., 1975) versus a generalized destruction of brain NE neurons (Amit & Sutherland, 1975/6), as well as use of animals selectively bred for ethanol preference in certain studies (Kianmaa et al., 1975), makes direct comparisons of results difficult. Since, in all cases, destruction of NE neurons did produce a change in ethanol intake, the existing evidence does indicate a role for NE neurons in determining quantitative aspects of its intake, but the nature of this interaction has yet to be clarified.

Administration of 6-OHDA can also produce a significant destruction of brain DA neurons (Ungerstedt, 1971), or may alter the function of DA neurons owing to destruction of inhibitory noradrenergic input (Antelman & Caggiula, 1977), and one may question whether the effects of 6-OHDA on ethanol intake are mediated by interference with the function of noradrenergic or dopaminergic neurons. Few investigations have been directed at specifically examining the role of DA neurons. Kianmaa et al. (1979) have attempted to selectively destroy the DA neurons that ascend from the brainstem to innervate the striatum and the mesolimbic areas of brain while leaving the hypothalamic DA terminals intact. The daily ethanol intake (measured in g/kg body weight) of the lesioned animals did not differ from that of sham-operated control rats. Studies by Davis et al. (1978) also question a role for DA neurons in ethanol self-administration. In these studies, animals were allowed to lever press for intragastric administrations of ethanol, and pretreatment of the animals with haloperidol (a DA receptor blocker) did not alter the pattern of lever pressing for ethanol. The studies of Kianmaa et al. (1979) and Davis et al. (1978) provide evidence that DA neurons do not play a primary role in the reinforcing effects of ethanol. However, the importance of DA neurons in the reinforcing properties of other abused substances (Wise & Routtenberg, 1983) prevents elimination of DA neurons from consideration in the context of ethanol's reinforcing actions.

Using an extensive range of ethanol solutions (3%–30%) in preference tests against water, Myers and Tytell (1972) have also implicated the serotonergic systems of brain as mediators of ethanol selection by animals. In these studies, the increase in brain serotonin (5-HT) levels by intraventricular

administration of the 5-HT precursor, 5-hydroxytryptophan (5-HTP), produced a diminution of ethanol intake. More recent studies by Rockman et al. (1979), utilizing inhibitors of 5-HT uptake (e.g., zimelidine), also demonstrate a decreased acceptance of ethanol solutions by animals. These authors postulate that increased levels of 5-HT in the synaptic cleft may be responsible for diminished intake.

Thus, several neuronal systems have been considered as mediators of the reinforcing properties of ethanol: the opiate, noradrenergic, dopaminergic, and serotonergic systems. Other neuronal systems have also been considered (e.g., cholinergic; Ho et al., 1975). However, since the bulk of current data implicates the catecholamine neurons as being of primary importance in mediating reinforcement, we will, in §6.3, concern ourselves with the examination of the effects of ethanol on the neurochemistry of NE and DA systems in animals acutely or chronically treated.

6.1.1b Is ethanol or are its metabolites the reason for drinking? To identify a compound responsible for producing a neuropharmacological effect, several criteria have to be satisfied. One criterion is that the administration of the suspected compound, in and of itself, would produce the pharmacological effect that is of interest. The second criterion would be that the compound should be present at the site of action at the time the effect is being witnessed. The exception to this second criterion would be the action of neurotoxins or related compounds that alter the function of the CNS in a rather permanent fashion.

A number of past and recent studies have attempted to ascertain whether the metabolic products derived from ethanol can alter ethanol preference. These products include acetaldehyde as well as the condensation products that can be formed from acetaldehyde, and the neurotransmitter catecholamines and indoleamines. In general, these studies have involved the *intracerebral* administration of the compound of interest prior to and/or during monitored periods of ethanol intake by animals in a free-choice situation. Myers and Veale (1969) investigated the effects of acetaldehyde on ethanol preference in rats and noted that ethanol ingestion could be increased by simultaneous injection of acetaldehyde into the brain. The doses of acetaldehyde they employed were 10 μ g every 15 minutes for the 5 days of the ethanol preference testing procedure. This dosing schedule can easily result in ventricular acetaldehyde concentrations of 2–5 mM, and such doses may act to release neuronal stores of catecholamines (Lindros, 1978). The released catecholamines may produce reinforcing consequences that could be associated with the intake of ethanol by the animal. Studies by Brown et al. (1979) have demonstrated that animals would learn an operant task to self-administer acetaldehyde directly into their cerebral ventricles. One may, however, pose the question of whether amounts of acetaldehyde comparable to those infused into the animal (Myers & Veale, 1969) or self-administered by the animal (Brown et al., 1979) are at any time present in the brain during or after oral ingestion of ethanol. The circulating levels of acetaldehyde are generally low in both animals (Sippel, 1974) and humans (Stowell et al., 1980) during ethanol

consumption, and a metabolic barrier protects the brain from being exposed to even these low levels of acetaldehyde. Numerous studies have demonstrated that levels of acetaldehyde in brains of ethanol treated animals are 10 to 20 times lower than levels of acetaldehyde found in the circulation (Sippel, 1974; Stowell et al., 1980; Tabakoff et al., 1976).

Several recent investigations may, however, bring additional perspectives of the issue, of brain levels of acetaldehyde in persons ingesting ethanol. Certain alcoholics with a recent history of heavy drinking (Lindros et al., 1980), and sons of alcoholic parents (Schuckit & Rayses, 1979), have been found to display significantly higher circulating levels of acetaldehyde after ethanol ingestion compared to non-alcoholic individuals. These findings indicate that in certain individuals, or in certain areas of brain, acetaldehyde may occur at levels higher than those expected from earlier studies. The higher estimates would still be well below the concentrations used to generate effects on ethanol preference, and one could conclude that the results generated by Myers and Veale (1969) and Brown et al. (1979) may have been a result of pharmacological effects of acetaldehyde, which would not be expected to occur under usual conditions of ethanol ingestion by animals or humans.

If significant levels of acetaldehyde were present in catecholamine and indoleamine neurotransmitter-rich areas of brain, one would expect formation of alkaloids derived from the condensation of serotonin with acetaldehyde (to form the beta-carbolines), and of dopamine or norepinephrine with acetaldehyde (to form the salsolinol and hydroxysalsolinol alkaloids) (Melchior & Collins, 1982). Acetaldehyde would also promote the condensation of dopamine with its own aldehyde derivative to form tetrahydroisoquinoline (TIQ) derivatives (Melchior & Collins, 1982). Experiments involving the intraventricular or site-specific injections of the above-mentioned alkaloids into the brains of rats or monkeys, and the testing of these animals for preference for solutions containing increasing concentrations of ethanol, have been performed (Myers, 1978; Myers & Hoch, 1979; Myers et al., 1982; Tuomisto et al., 1972). The major finding of these studies is that animals injected with salsolinol, tetrahydropapaveroline (THP), or beta-carbolines will ingest solutions containing higher concentrations of ethanol and will thus increase their daily consumption. The duration of action of these alkaloids (up to 10 months after intraventricular administration) indicates that they produce rather permanent changes in some yet unknown controller of ethanol intake (Myers & Melchior, 1977). Although several groups have generated data indicating that chronic infusion of THP and beta-carbolines during the testing of animals' preference for ethanol increases the acceptance of ethanol, results on the effects of single administrations of the alkaloids have not been consistent (Duncan & Deitrich, 1980; Myers & Oblinger, 1977). Amit et al. (1982) have summarized and discussed the negative findings. A major aspect of the methodology of the experiments that report an increased intake of ethanol by animals treated with the beta-carboline and TIQ alkaloids is that the animals are infused continuously with the alkaloids during the preference testing procedure. If the alkaloids activate brain-reward pathways, or are otherwise reinforcing, one might expect that, as with acetaldehyde, a positive association

could develop between ethanol selection and the rewarding effects of the infused substance. Such learned behavior could also explain the long-lasting effects of TIQ administration. Only one study has, to date, directly addressed the question of the reinforcing effects of TIQs, and this study has provided preliminary evidence that rats will lever press for intraventricular infusions of salsolinol (Amit et al., 1982).

If one conjectures that the TIQ alkaloids are generated from acetaldehyde produced during the intake of ethanol by animals or humans, then one would suspect that the ingestion of large doses of ethanol would result in the *in vivo* production of TIQ alkaloids. Sophisticated techniques of analytical chemistry (GC-MS, radioisotope studies) have been applied to this problem and have produced mostly negative results (Bloom et al., 1982). One notable exception is the demonstration of significant quantities of salsolinol in brains of human alcoholics, and in rats forced to consume ethanol for extended periods of time (Sjoquist et al., 1982a,b). However, Sjoquist and co-workers also found that brains of humans who had not ingested large quantities of ethanol, and brains of rats that had not consumed any ethanol, contained significant levels of salsolinol. It is of interest that the higher levels of salsolinol were noted in dopamine-rich brain areas, and we have already mentioned that dopaminergic neurons have been considered to play a major role in control of brain-stimulation reward systems (Wise & Routtenberg, 1983).

If one were to consider gathering neurochemical evidence to support the reinforcing role for the TIQs, one would examine the effects of these alkaloids on the catecholamine neurons of brain, or on other neuronal systems (e.g., opiate) that may be involved in reinforcement at the neurochemical level. A substantial amount of such work has been completed, and this work has been recently reviewed (Melchior & Collins, 1982). The structural similarities between the TIQ alkaloids and the catecholamines promoted investigations on the effects of these alkaloids on uptake, release, and receptor binding properties of the neurotransmitter catecholamines. Both salsolinol and THP affect all of the above-mentioned processes that govern the actions of the catecholamine neurotransmitters (Melchior & Collins, 1982), but the potency of these alkaloids is well below what would be necessary to consider any of these actions as being critical for mediating the effects of the alkaloids at concentrations found to be present *in vivo*. The actions of salsolinol and THP on opiate receptor binding properties have also been examined, as have the actions of the beta-carbolines on the binding properties of the benzodiazepine receptors (Melchior & Collins, 1982). Although certain beta-carbolines are potent ligands at the benzodiazepine receptors of brain, the structural features characterizing the more potent beta-carbolines would not be expected to be generated by way of *in vivo* condensation of acetaldehyde and the indoleamine transmitters. The TIQ alkaloids are poor ligands for the opiate receptors of brain (Melchior & Collins, 1982). Thus, a cursory examination of the TIQ and beta-carboline alkaloid pharmacology provides little evidence regarding the neurochemical systems through which such alkaloids may produce their reinforcing effects and/or their effects on intake of ethanol.

On the other hand, if one examines the quantities of the alkaloids injected

into the ventricles of animals used for studies of alcohol preference, one finds that local concentrations well within the μM - mM range, would be present at the time of alkaloid infusion (Myers & Melchior, 1977; Myers, 1978). On the basis of this observation, one might well expect pulsatile pharmacological effects of the alkaloids on the catecholamine or other systems of brain. One might conclude that these pharmacological effects may be centred around the binding of the alkaloids (salsolinol and THP) to opiate, beta-adrenergic, and/or dopamine receptors in brain (Melchior & Collins, 1982); and altered function of the opiate, NE, and DA systems could, in turn, be perceived by the animal as the rewarding consequence of ethanol selection.

Overall, the contention that *in vivo* formation of the TIQ or beta-carboline alkaloids due to ethanol consumption is responsible for development of ethanol dependence, or for increasing ethanol preference, currently has little experimental support. Nevertheless, the pharmacological effects of the *exogenously* administered TIQ alkaloids may well produce an increase in an animal's preference for ethanol in a free-choice situation, and one can contend that such preference is a result of development of an association between the reinforcing actions of the alkaloids and the ethanol offered in the free-choice paradigm.

6.1.2 Other Factors Related to Initial Preference for Ethanol

6.1.2a Is an animal's initial sensitivity to the intoxicating effects of ethanol related to its consumption of ethanol? Even a cursory examination of the range of ethanol consumption by humans and animals indicates the vast heterogeneity in individual patterns of consumption and quantities consumed. We will not dwell on the studies that have demonstrated that ethanol consumption, at least in animals, is determined by both genetic and environmental variables (see Belknap, 1980, for review). The demonstration of a genetic determinant of ethanol preference or aversion in animals has presented an opportunity to assess which of the other ethanol-related phenomena may be closely linked to or may be determinants of ethanol preference. If one selectively breeds animals for ethanol preference and aversion, one can assume that, by serial selection, one would approach a condition in which the lines differ maximally at all gene loci that contribute to determining ethanol preference, but phenotypes under the control of non-relevant loci should not differ between the lines. Thus, comparison of selectively bred high- and low-response lines can be very useful in the study of factors contributing to the determination of a particular phenotype. Two lines of rats selectively bred for ethanol preference and one line of rats bred for CNS sensitivity to administered ethanol have been used to ascertain the relationship between ethanol preference and the susceptibility to intoxication (Eriksson, 1969; Lumeng et al., 1977; Rusi et al., 1977), and the results of these studies are discussed below. In addition, mice from inbred strains with a known history of ethanol preference (Rogers, 1972) have been used to ascertain sensitivity to the intoxicating effect(s) of ethanol. This latter approach provides for a correlative analysis between an animal's preference for ethanol and its response to administered ethanol. The rationale for most studies of preference for and sensitivity to ethanol was based on the desire to understand whether animals ingest or avoid

it because of the experienced pharmacological effects. Many of the studies of the response of ethanol-preferring or ethanol-avoiding animals to administered ethanol have, however, used high doses that produce behavioral effects never noted under conditions where an animal is voluntarily consuming ethanol. A researcher may consider that all effects of ethanol on the CNS are described by monophasic, linear dose-response curves, and any effect in animals differing in sensitivity would be described by a parallel series of such curves. Under such conditions, the dose used in a particular study would have little bearing on the interpretation of results. However, there is ample evidence that the low- and high-dose effects do not form an uninterrupted or linear continuum (Pohorecky, 1977), and care has to be exercised in conjecturing a relationship between the sensitivity of an animal to the hypnotic or any other high-dose effect and ethanol preference.

A more informative examination would concern an animal's sensitivity to lower doses that produce blood ethanol levels comparable to those attained during voluntary consumption. The behavior most often examined after administration of low doses (in the range of 1–2 g/kg) to mice or rats is the animal's locomotor activity. The assumption has been made that the locomotor activation produced by low doses reflects the positive reinforcing effects of ethanol, but examination of data on these activating effects indicates that the effects are most prominent in strains of mice that avoid ethanol (Kianmaa & Tabakoff, 1982). Certain lines of rats bred for ethanol avoidance also seem to be more sensitive to the incoordinating or sedative effects (Waller et al., 1983). Such data would lead to the hypothesis that animals bred to prefer ethanol are, in general, more resistant to its overt behavioral effects. The demonstration of the converse of this relationship would certainly strengthen the link between ethanol preference and an animal's initial sensitivity to the behavioral effects. However, at least two attempts at establishing this relationship have yielded negative results (Collins, 1981; Riley et al., 1977).

6.2 CHARACTERISTICS OF FACTORS THAT CAN ALTER THE INITIAL LEVEL OF PREFERENCE FOR ETHANOL

In the following section we describe studies on tolerance development from which one can conclude that: (1) development of functional tolerance is genetically modulated and thus has biological determinants; (2) development of tolerance under certain conditions can be considered to be a specific instance of learning, whereas under other conditions tolerance development may reflect neuroadaptive processes not normally associated with learning; and (3) tolerance does not develop in an equivalent fashion to all actions of ethanol, and the initial balance between systems that control the positively reinforcing and the aversive consequences of its intake can be significantly altered by development of tolerance.

6.2.1 *Typology of Tolerance*

Both the experimental paradigms and the data related to investigations of ethanol tolerance have been extensively reviewed (Tabakoff et al., 1982). There is no doubt that tolerance does develop to certain actions of ethanol, but

a close examination of the experimental designs indicates a paucity of data in a number of areas, and reevaluation of past work also indicates that factors as yet poorly defined may interact to control the development of tolerance. The most commonly measured effects of ethanol in relationship to development of tolerance have been ethanol-induced sedation and incoordination. Studies are usually constructed such that the initial effects of ethanol are measured and the animal then consumes or is administered ethanol for a period of time. The animal's behavior during the chronic administration period, or in response to a subsequent test dose of ethanol, is taken as a measure of tolerance. Depending on the concerns of the experimenter, a change in the response of the animal to ethanol may then be related to a further behavioral, physiological, or biochemical consequence of its chronic administration.

Although it is well known that the overt expression of tolerance can reflect both functional and metabolic (dispositional) components, seldom do studies of tolerance reveal a concerted attempt to distinguish between the relative contributions of these components to the observed results. If tolerance is monitored by changes in the duration of the ethanol effects, rather than by the extent of an effect at particular tissue levels of ethanol, one has to consider that metabolic tolerance (i.e., increased elimination rate) could be responsible for a diminished duration of effects. These considerations become critical in studies aimed at elucidating the neurochemical determinants of tolerance. Certainly, little use can come from an examination of brain mechanisms when the factor responsible for tolerance is located in the liver.

The paradigm within which ethanol is chronically administered to an animal is also important in determining what role classical conditioning may play in the development of tolerance. In the course of repeated administration, a conditioned response may develop in association with those environmental stimuli that have been paired with the unconditioned pharmacological response (i.e., the effect of ethanol). This conditioned response has, in a number of instances, been shown to represent a homeostatic adaptation to compensate for the physiological perturbation produced by the drug (Hinson & Siegel, 1980). The conditioning model proposes that the development of such a compensatory response eventually attenuates the direct pharmacological action of the drug, and that the observed result of such attenuation is tolerance. Studies of this type of tolerance have utilized repeated ethanol administrations within a consistent, predictable environment and demonstrated that tolerance could be evidenced only in the environment with which the drug had been paired. If ethanol was administered outside of that environment, the organism could not be demonstrated to be tolerant. We have labelled such conditioned tolerance as "environment-dependent" (Tabakoff et al., 1982) to distinguish it from tolerance that develops in paradigms in which learning or conditioning would be expected to play but a minor role. "Environment-independent" tolerance has been shown to develop under conditions where ethanol is administered as part of a liquid diet or by inhalation techniques, and responses to ethanol are tested after its intraperitoneal (i.p.) administration in a novel environment (e.g., Ritzmann & Tabakoff, 1976). Examination of experimental protocols of studies concerned with environ-

ment-dependent and environment-independent forms of ethanol tolerance indicates that continuous administration of significantly higher amounts of ethanol is necessary to produce environment-independent tolerance, as compared to amounts necessary to produce environment-dependent tolerance (Melchior & Tabakoff, 1981; Ritzmann & Tabakoff, 1976). One may consider that the strength of stimulus for CNS adaptation may have to be increased when other contingencies determining an animal's response to ethanol cannot come into play. However, environment-dependent and -independent components of tolerance may be determined by quite distinct mechanisms that develop in parallel, as do the metabolic and functional aspects of tolerance. The repertoire of adaptive responses of the CNS to the presence of ethanol is not yet defined, but one should clearly be cognizant of the fact that the experimental model one uses to establish the behavioral concomitants of tolerance will certainly influence the result of any study aimed at determining the biochemical determinants of tolerance.

6.2.2 Does Tolerance Develop to All Behavioral and Physiological Effects of Ethanol?

The experimental protocols for studying the development of tolerance in animals in most cases concentrate on only a single consequence of ethanol's actions. The studies that have monitored more than one effect, however, indicate that tolerance develops at different rates to the various effects, and, in fact, certain actions of ethanol may not demonstrate the development of tolerance. For instance, when mice were fed ethanol incorporated into a liquid diet, and ethanol-induced hypothermia and loss of righting reflex were measured prior to and subsequent to periods of consumption, tolerance to the hypothermic effect was shown to develop more rapidly than tolerance to the hypnotic effect (Ritzmann & Tabakoff, 1976; Tabakoff & Ritzmann, 1977).

Low doses of ethanol have been demonstrated to produce locomotor activation in certain strains of mice (McClearn & Anderson, 1979). When DBA and BALB mice demonstrating such locomotor activation were fed ethanol in a liquid diet, no development of tolerance was evidenced to this effect (Kiiianmaa & Tabakoff, 1982). However, these same animals demonstrated significant tolerance to the hypnotic effects of high doses (Kiiianmaa & Tabakoff, 1982). Studies by Masur and Boerngen (1980), using Swiss-Webster mice, indicated similar results. These authors administered daily doses of ethanol by i.p. injection and noted that tolerance to its sedative effects developed during the month-long experiment. Interestingly, as animals became tolerant to the sedative effects, locomotor activating effects could be distinguished, and the activating effects progressively increased as the sedative effects decreased. These authors concluded that the development of tolerance to the sedative effects of ethanol allows for expression of its activating effects.

Studies on the development of tolerance to the overt behavioral and physiological effects of ethanol indicate a differential propensity for various functions to develop such tolerance. An important action of ethanol, in the context of this review, is its reinforcing effect, but experimental work has not been directed at answering the question of whether tolerance develops to its

reinforcing properties. Little evidence has been found for tolerance development to the positively reinforcing properties of several other abused substances (Schuster, 1978). However, the biochemical mode of action of ethanol on neuronal systems deemed to be important in reinforcement differs from that of other abused drugs, and a modicum of evidence exists from studies of brain self-stimulation (see above) that tolerance does develop to certain effects of ethanol on the systems mediating reinforcement. Biochemical studies, which will be discussed below, also indicate alterations in the effects of ethanol on neuronal systems important for reinforcement after its chronic feeding to an animal, but the basic question regarding the development of tolerance to its reinforcing effects remains essentially unanswered. The evidence for differential rates of development of tolerance to the various other actions of ethanol does allow for the following conjecture. Ethanol intake could be assumed to be controlled by the balance of its positively reinforcing and aversive properties, or by physiologically derived cues of its action that become associated with particular expectations of effect (see §8). Differential rates of tolerance development to various physiological effects of ethanol, including the aversive and reinforcing properties, would alter the quantitative aspects of its consumption. Tolerance development to its aversive properties or its incapacitating properties would lead to an increased ability to experience its positively reinforcing properties. For instance, when one examines the neurochemical effects of ethanol on systems postulated to mediate reinforcement (e.g., DA systems; see below), one finds that relatively high doses are necessary to activate these systems. Tolerance to the sedative effects would allow for attainment of sufficiently high ethanol levels for their activation. Karoly et al. (1978) demonstrated that monkeys trained to self-inject ethanol maintained blood ethanol levels of 400 mg% during 6-hour periods of access to ethanol. Certainly, tolerance to its hypnotic effects must have been present in these animals to allow them to carry on the self-administration procedure. Under conditions of unlimited, 24-hour-a-day access to ethanol, monkeys will self-administer large amounts over several days, will become severely intoxicated, and will then cease or significantly reduce the self-administration (Winger & Woods, 1973). One wonders whether tolerance developed to the reinforcing properties of ethanol, and whether diminished reinforcement led to termination of self-administration, or whether the aversive consequences of severe intoxication overrode the positively reinforcing properties. Since similar patterns of ethanol intake and self-imposed abstention have been noted in humans (Mello & Mendelson, 1972), the factors mediating such behavior are apparently important.

6.2.3 *Genetics of Tolerance Development*

Genetic determinants for the development of acute and chronic tolerance to the sedative/hypnotic effects of ethanol have been recently demonstrated. Using inbred mice of the C57BL and DBA strain, we found that C57BL mice developed acute tolerance to the hypnotic effects of ethanol, but DBA mice did not; specifically, the C57BL mice regained their righting reflex at significantly higher brain ethanol levels than those at which they lost the

righting reflex, whereas DBA mice lost and regained their righting reflex at similar brain ethanol levels (Tabakoff & Ritzmann, 1979). Strain-dependent differences in the development of what may be considered acute tolerance were also demonstrated by Grieve et al. (1979). These workers administered ethanol continuously by inhalation and demonstrated that the significant tolerance in C57BL mice developed within 2.5 hours, whereas no tolerance was evident in DBA mice maintained under equivalent conditions.

Neither SS nor LS mice demonstrated evidence for development of acute tolerance to the hypnotic effects of ethanol; thus, the major differences in "sleep-time" between the SS and LS lines of mice given a single dose have to be totally attributed to differences in genetically determined initial CNS sensitivity to ethanol (Tabakoff & Ritzmann, 1979). However, studies in our laboratory on *rates* of development of *chronic* tolerance in SS and LS mice showed that SS mice developed chronic, functional tolerance to the hypnotic effects faster than LS mice (Tabakoff et al., 1980).

The rapidity with which tolerance develops is determined not only by the genetics of the individual, but also by prior cycles of tolerance development. DeSouza-Moreira et al. (1981) and Kalant et al. (1978) have demonstrated that an initial cycle of tolerance development in mice or rats may "prime" the adaptive machinery of an animal, such that tolerance can develop at a more rapid rate during subsequent administration of ethanol. In these experiments, sufficient time was allowed between sessions of ethanol administration for the overt sensitivity of the animals to return to the initial pre-administration baseline levels. A similar phenomenon has been described in human alcoholics. Mendelson and La Dou (1964) noted that individuals participating in studies involving consumption of alcohol could attain a high level of consumption faster if they had a prior (recent) history of tolerance development.

One can conclude that if a negative feedback on the quantitative aspect of ethanol consumption is exerted by factors to which tolerance can develop, the genetic makeup and prior tolerance history of individuals can promote the loss of the negative feedback control by accelerated tolerance development. The result would be expected to be greater intake of ethanol by the individual.

6.3 NEUROCHEMICAL ACTIONS OF ETHANOL THAT MAY INFLUENCE ETHANOL CONSUMPTION

6.3.1 Acute and Chronic Effects of Ethanol on Neuronal Systems That Generate the Perception of Reward

The neuronal systems that will come under scrutiny in this section are primarily the noradrenergic and dopaminergic systems of brain. These networks of neurons certainly do not act in isolation in the processes of integrating and transmitting information, and one has to clearly keep in mind the caveat that the observed effects of ethanol may be a result of its actions on other neuronal systems that in turn influence the activity of the systems we will have under consideration. It is also clear that neuronal systems other than noradrenergic and/or dopaminergic systems participate in sensory and motor functions related to perception of, and response to, rewarding stimuli. The DA and NE

systems were selected since sufficient information exists that these neuronal networks may function in the experience of reward (Fibiger, 1978; Wise & Routtenberg, 1983), and a certain amount of already-discussed experimental evidence indicates the involvement of these systems in mediating the reinforcing effects of ethanol.

6.3.1a The effect of ethanol on NE neuron function. The neurochemistry of ethanol's action on brain noradrenergic networks has been studied *in vivo* after ethanol administration by measuring: the absolute levels of NE; the rate of synthesis of NE from radioactively labelled precursors; the rate of decline of NE levels after synthesis inhibition; the accumulation of the NE precursor, DOPA, which is produced by the rate-limiting enzyme (tyrosine hydroxylase) in the pathway of NE synthesis; and the levels of the metabolites of NE present in brain and CSF. All of these studies have been aimed at ascertaining the amount of the neurotransmitter being released from NE neurons after ethanol administration, with the implicit assumption that an increase in synthesis and metabolites of NE indicates increased release.

Investigations of rates of NE synthesis and release in whole brain have produced indications of a biphasic effect of ethanol. At lower doses (Bacopoulos et al., 1978; Corrodi et al., 1966), and soon after administration of high doses (Hunt & Majchrowicz, 1974), NE turnover is increased. When circulating levels of ethanol have reached peak values after the administration of high doses, NE turnover is depressed (Hunt & Majchrowicz, 1974; Pohorecky, 1974).

Ethanol-induced *release* of NE from the NE-containing neurons can be indicated by studies that monitor the rates of decline of NE after administration of ethanol in conjunction with the inhibition of NE synthesis by drugs such as alpha-methyl-p-tyrosine (alpha-MT). The studies that monitor the levels of the NE metabolites can also be used in this context. Examination of the results of such studies with animals indicates a stimulatory action of low and intermediate doses or low and intermediate (about 300 mg%) levels of ethanol on release of NE (Corrodi et al., 1966; Karoum et al., 1976), and an inhibitory effect of high, anesthetic levels on NE release (Pohorecky, 1974). Interestingly (though the studies should be interpreted with certain caveats in mind; Davis et al., 1967), measurement of the NE metabolite, MHPG, in the CSF of intoxicated humans (alcoholics and nonalcoholic volunteers) indicated an increased release of NE in the CNS (Borg et al., 1981) after ingestion of 1–1.5 g/kg ethanol.

Studies with animals, in which anatomically specific changes in NE metabolism were monitored after administration of ethanol, have provided additional support for the assertion that lower doses increase release of NE, particularly in certain brain areas, and have led to the conclusion (Bacopoulos et al., 1978) that the altered NE metabolism in the brainstem is primarily a result of altered activity of the diffuse NE network of the brainstem reticular activating system or the descending NE neurons that synapse in the brainstem.

From the currently available neurochemical studies of NE metabolism, one can conclude that sedative and anesthetic doses of ethanol significantly

diminish the activity of most, if not all, of the brain's NE neurons. Lower doses (at or below 2 g/kg), which have, in certain experiments, been used to demonstrate ethanol-induced locomotor activation (Kianmaa & Tabakoff, 1982; McClearn & Anderson, 1979), activate selectively the NE neurons of the reticular activating system and the descending (motor) pathways. The dose threshold for ethanol's effects on the metabolism of NE, and, presumably, the activity of brainstem NE neurons, is unknown. If doses as low as those used in studies on brain-stimulation reward (i.e., 0.5 g/kg) could alter the activity of NE neurons of the reticular activating system, one could conjecture that the increased responding witnessed in the brain stimulation studies was a result of the increased arousal produced by the activation of the NE component of this brainstem system. The effects of ethanol, given at doses of approximately 1.0 g/kg, on CNS NE metabolism in humans (Borg et al., 1981) may indicate that the NE brainstem networks of human brain are activated, as are these networks in animals. In addition, Engel and co-workers have demonstrated that administration of alpha-MT to human subjects prior to their imbibing ethanol significantly diminishes the behavioral activation produced by ethanol (Ahlenius et al., 1973). Alpha-MT is an inhibitor of NE synthesis, and the depletion of NE stores in neurons prior to ethanol administration would be expected to diminish the amount of NE available to be released by ethanol.

If one contends that the descending NE neurons of brain, or the NE neurons in the brainstem reticular activating system, directly mediate ethanol reinforcement, one could say that moderate doses activate these systems, and this would be a reason for drinking. There is little evidence to support such a role for these brainstem NE neurons, however, and such a contention is inappropriate. One should consider that the activation of brainstem NE networks may rather mediate an increased access of external stimuli to the higher brain centres and allow for the behavioral activation produced by the low to moderate doses of ethanol. This event could also be considered as a discriminative stimulus.

The *chronic* ingestion of ethanol has been demonstrated to increase the ethanol concentration range over which the neurochemical measures of NE neuron activity indicate an activation of brain NE systems. A variety of indirect measures of NE neuron function (Hunt & Majchrowicz, 1974; Karoum et al., 1976; Pohorecky, 1974) indicate an increased activity of the NE neurons of rat brain, even at blood ethanol levels in excess of 300 mg% (Pohorecky, 1974). Intoxicated humans admitted to a detoxication clinic were also found to have significantly elevated CSF levels of the NE metabolite MHPG (Borg et al., 1981). Their blood ethanol levels ranged from approximately 100 to well over 400 mg%, and the CSF MHPG levels were positively correlated with the individual's blood ethanol levels. Thus, one can conclude that the inhibitory effects of high concentrations of ethanol on brain NE neurons produced by a single dose are diminished after chronic ingestion by animals or humans (i.e., tolerance develops). The available data do not provide information for an anatomical analysis of which NE systems become more resistant to the inhibitory effects of ethanol. One may consider that chronic ingestion produces a shift to the right of the dose-response curve for the inhibitory effects of

ethanol on NE neurons in general, but no direct neurochemical data are available to indicate whether it also produces a shift in the threshold for activation of NE neurons. With regard to behavioral sequelae of chronic administration, evidence gathered from studies of the effects of ethanol on locomotor activity and bar pressing for brain stimulation indicates that the behavior-inhibiting effects of higher doses are reversed, whereas behavior-activating effects of low doses are unaltered or possibly even increased (Magnuson & Reid, 1977) by prior chronic ingestion of high doses. The developed tolerance within the NE neurons or associated systems would produce neuronal resistance to the deactivating effects of ethanol and could allow for more substantive expression of its activating effects on NE neurons and NE-mediated behaviors.

6.3.1b Effects of ethanol on DA neuron function. It has been argued that increased activity of striatal DA neurons may be of greater importance than activity of NE neurons for the increase in locomotor behavior produced by low doses of ethanol (Carlsson et al., 1974; Strombom & Liedman, 1982). The mesolimbic DA neurons seem to be significantly more important than the nigro-striatal DA system in initiating increases in locomotor activity produced by ethanol in rodents (Bannon & Roth, 1983; Liljequist & Engel, 1979), and the mesolimbic DA system has recently also been implicated in mediating the reinforcing effects of opiates and cocaine (Goeders & Smith, 1983; Wise & Routtenberg, 1983). Most studies of ethanol's actions on the DA systems, however, have been concerned with its neurochemical effects on the nigro-striatal DA system. For a number of reasons (Bannon & Roth, 1983), one cannot accurately extrapolate from effects witnessed in the nigro-striatal DA neurons to generate solid hypotheses regarding the actions of ethanol on the mesolimbic DA neuronal network. The data discussed below are therefore presented mainly to illustrate certain principles that may be, in general, important in describing the neurochemical actions of ethanol on the DA systems of brain.

There is consensus that acute doses of ethanol can produce an "uncoupling" of stimulus-induced release of DA from the concomitant changes in synthesis (Bustos & Roth, 1976; Gysling et al., 1976). This phenomenon can explain the divergent interpretations of ethanol's effects on the functional state of the DA neurons of brain. If one monitors the actions of ethanol on striatal synthesis of DA in mice or rats, one notes a dose-dependent increase in DA synthesis. The increased *synthesis* of DA is evident even at low doses (about 2 g/kg) (Carlsson & Lindqvist, 1972; Fadda et al., 1980; Kianmaa & Tabakoff, 1983), but it is clear that the animal's sensitivity to this effect is genetically determined (Kianmaa & Tabakoff, 1983). Measures of DA *release* also demonstrate the genetically determined sensitivity to ethanol in mice, and there is a biphasic effect on this neurochemical parameter (Kianmaa & Tabakoff, 1983). Low doses (about 2 g/kg) *diminish* or do not alter the release of DA in the striatum, whereas higher doses (about 3 g/kg) increase the release of DA and the accumulation of DA metabolites. This general trend is also evident in studies performed with rats (Bacopoulos et al., 1978; Fadda et al., 1980; Karoum et al., 1976).

The decrease in DA release in rat and mouse brain witnessed at doses ranging from 0.8 to 2.0 g/kg is of interest with regard to studies that measured the effect of ethanol on brain-stimulation reward and on conditioned place preference. In the brain-stimulation reward experiments (Carlson & Lydic, 1976), ethanol doses of 0.9 and 1.2 g/kg increased the threshold for current to elicit self-stimulation, and one wonders if this phenomenon may not be related to ethanol-induced diminution in DA release from the DA neurons, which may be important for mediating brain-stimulation reward from the lateral hypothalamus. The diminution of DA release produced by ethanol doses in the range of 1 g/kg (i.p.) may also have played a role in the conditioned aversion that was demonstrated with rats given such doses in a place preference experiment (van der Kooy et al., 1983). If one considers the activity of DA neurons of brain to be of significance in generating the reinforcing properties of drugs' (Wise & Routtenberg, 1983), one has to consider that ethanol effects are clearly dose-dependent and modulated by genetically determined factors. In general, low doses of ethanol (about 0.5 g/kg in mice and rats) have no effect on DA release, moderate doses (0.8–2.0 g/kg) have no effect or *decrease* release, and only high doses (about 2.5 g/kg) increase release. These effects have been demonstrated predominantly in the striatum, and, as already mentioned, the extent of the effect is determined by the genetic constitution (strain) of the animal one examines. The DBA and BALB mice, for instance, are significantly more sensitive to the inhibitory effects of ethanol on DA release compared to C57BL mice (Kiianmaa & Tabakoff, 1983). Pursuing the currently popular line of thinking regarding the activity of DA neurons and reinforcement, one would conclude that low to moderate doses of ethanol, which inhibit DA release in brains of the DBA and BALB mice, would block reinforcement in these animals. These animals do avoid the intake of ethanol in a free-choice situation against water. The C57BL mice, which consume significant amounts of ethanol in a free-choice paradigm (Rogers, 1972), are less sensitive to the inhibitory effects of lower doses on DA metabolism (Kiianmaa & Tabakoff, 1983).

High doses of ethanol stimulate DA release in both mice and rats (Fadda et al., 1980; Karoum et al., 1976; Kiianmaa & Tabakoff, 1983), but these doses will usually incapacitate animals that do not possess a genetically determined resistance to, or have not developed tolerance to, the sedative effects. One may expect that only animals with an inherent or *acquired* resistance to those sedative effects would be able to attain, by self-administration, the levels of ethanol necessary to activate the release of DA. The necessity of achieving high circulating levels of ethanol to stimulate DA release may be responsible for the pattern of intake demonstrated in the studies of Karoly et al. (1978), wherein the dependent monkeys, self-administering, maintained blood ethanol levels of approximately 400 mg %.

The chronic intake of high levels (about 9 g/kg/day) by an animal not only produces tolerance to the sedative effects but also eventually diminishes the effectiveness with which ethanol can stimulate the release of DA (Barbaccia et al., 1982; Kiianmaa & Tabakoff, 1983). The time course of tolerance development for the hypnotic versus the DA-releasing properties of ethanol could be of

importance for determining the balance between factors that may be promoting or curtailing intake, but these time courses have not been detailed. It is also evident that the intake of high doses of ethanol would have aversive consequences in addition to sedation, and diminution of its positive reinforcing properties (e.g., by development of tolerance to its DA-releasing properties) would allow these aversive consequences to predominate and would curtail intake.

In summary, relatively high concentrations of ethanol are necessary to enhance DA release in the striatum, and even higher levels, or a period of priming with ethanol, may be necessary to produce this effect in the frontal cortex. To reach these levels, an animal has to traverse a portion of the dose-response curve in which ethanol may diminish the release of DA. If DA neurons mediate the reinforcing properties of ethanol, the diminished DA neuron function would allay reinforcement. Certain animals, however, have a genetically determined resistance to the inhibitory effects of ethanol on DA release. The chronic intake of high doses produces a tolerance to its DA-releasing effects, and thus reinforcement would be diminished in the chronically treated animal unless greater quantities were ingested by the tolerant subjects.

6.3.2 Molecular Mechanisms by Which Tolerance Is Produced

6.3.2a Studies to determine the neurobiological systems that may be responsible for changes in tolerance. It is important to differentiate, both semantically and experimentally, between systems that are important in facilitating or "priming" changes in tolerance, and those systems in whose function ethanol tolerance actually resides (systems important for "expression" of tolerance). The possible influence of associative (e.g., Pavlovian conditioning) factors in the development of tolerance has led to investigations of what effects pharmacological manipulations known to disrupt memory consolidation might have on the development of tolerance.

In animals, ablation of the frontal cortex and treatment with the inhibitor of protein synthesis, cycloheximide, have each been shown to inhibit the development of tolerance (LeBlanc & Cappell, 1977). Both of these procedures have previously been shown to interfere with consolidation of learned responses.

Several of the neurotransmitter systems of brain (e.g., 5-HT and NE) have also been demonstrated to play an integral role in memory consolidation processes, and the effects of selective destruction of these systems have also been examined with respect to ethanol tolerance development.

The *development of tolerance* to ethanol in rats was slowed by chronic administration of p-chlorophenylalanine (pCPA) in a dosage regimen that produced and maintained approximately 95% depletion of brain serotonin (Frankel et al., 1975). Similarly, selective destruction of serotonergic neurons with the neurotoxin, 5,7-dihydroxytryptamine, significantly slowed tolerance development (Lê et al., 1980). Tolerance to both the hypothermic and motor-impairing effects of ethanol was evaluated in these studies. Once tolerance was established, pCPA did not affect its expression, but it did increase the rate of loss of tolerance. Thus, part of the inhibitory effect on tolerance development

could have been due to the accelerated loss of tolerance during the tolerance-induction phase.

Partial destruction of mouse brain catecholamine systems with the selective neurotoxin, 6-hydroxydopamine (6-OHDA), also blocked the development of tolerance to the hypothermic and sedative effects of ethanol in studies where mice were fed an ethanol-containing liquid diet (Tabakoff & Ritzmann, 1977). In these studies, the rate of acquisition of tolerance was not determined, so that a decrease in the rate of tolerance development cannot be ruled out. However, in a paradigm that produced environment-dependent tolerance, 6-OHDA treatment was shown to slow tolerance development (Melchior & Tabakoff, 1981). In all studies with 6-OHDA, noradrenergic systems, rather than dopaminergic systems, were postulated to be of importance for development of ethanol tolerance, and the destruction of noradrenergic systems after tolerance had developed did not affect expression of this tolerance (Melchior & Tabakoff, 1981; Tabakoff & Ritzmann, 1977).

Additional evidence of similarities between neurobiological processes that modulate memory and ethanol tolerance has recently been obtained with the use of vasopressin and related peptides. Administration of vasopressin, which has previously been shown to prevent extinction of learned behaviors (van Wimersma Greidanus et al., 1983), to mice chronically treated with ethanol, postponed the disappearance of ethanol tolerance after termination of ethanol administration (Hoffman et al. 1979).

The above-described studies on the effect of neurotoxins on the *development* of tolerance (Lé et al., 1980; Tabakoff & Ritzmann, 1977) also bring out the important fact that the presence of ethanol in the milieu of neuronal systems is a necessary, but not sufficient, factor for the development of tolerance. The appropriate activity of certain neuronal systems (e.g., noradrenergic neurons), and the interaction of the various neuronal systems in the presence of ethanol, seem to be controlling factors for tolerance development, at least in animals.

6.3.2b Studies of the molecular determinants of ethanol tolerance. To elucidate the biochemical systems that actually become tolerant to ethanol (i.e., to identify systems in which tolerance actually resides), investigators have utilized the concept that tolerance (resistance to ethanol) should be demonstrable in systems that are initially inhibited or disrupted by the presence of ethanol. The neuronal membrane contains entities that modulate ion permeabilities and recognize transmitter molecules, and these entities are important for conduction and transmission of information in the CNS. Ethanol has been shown to affect ion permeabilities in excitable tissue (Seeman, 1972), and ethanol added to neuronal membranes has been shown to produce a disordering effect (increased "fluidity") in such membranes. However, neuronal membranes derived from ethanol-fed mice were found to be resistant to such a disordering effect, and this resistance (tolerance) has been postulated to be the result of increased cholesterol levels or changes in phospholipid composition in the membranes of the ethanol-tolerant animals (Goldstein & Chin, 1981). Cholesterol interacts with the hydrocarbon chains of the phospholipid in the

neuronal membranes, and changes in either the hydrocarbon chain saturation per se or membrane cholesterol content would markedly affect fluidity of neuronal plasma membranes. Such changes could impart to the membrane a resistance to ethanol's effects.

The lipids of the neuronal membranes are its major structural components, but the major functional components (i.e., ionophores, receptors, and transport enzymes) are proteinaceous in nature. Changes in membrane lipid composition can, however, alter the activity of these membrane-bound functional proteins (Tabakoff & Hoffman, 1983). For instance, the functions of neurotransmitter-sensitive adenylate cyclase (AC) and the sodium/potassium-activated ATPase have both been shown to be sensitive to changes in membrane lipids, and the activity of both of these enzymes has been shown to be altered by chronic ethanol administration (see references in Tabakoff & Hoffman, 1983). Thus, it may be postulated that chronic ethanol administration produces adaptive changes in the lipids of neuronal membranes, and the changes in lipids may in turn alter the function of certain enzymes and receptors residing in the neuronal membranes. Changes in the activity of the functional components of the neuronal membranes and their resistance to ethanol's actions would contribute to the expression of the ethanol-tolerant state. The postulated changes in the lipids of neuronal membranes provide a testable hypothesis regarding the determinants of ethanol tolerance, but one has to recognize the fact that this is not the only possible explanation of tolerance development. For further discussion of the acute and chronic molecular effects of ethanol on the CNS, the reader is referred to recent reviews (Tabakoff & Hoffman, 1980, 1983).

7. APPLICATION OF THE DATA AND THE HYPOTHESIS TO UNDERSTANDING PATHOLOGICAL DRINKING PRACTICES AND CONSEQUENCES OF THOSE PRACTICES

A person's (or animal's) initial experiences with ethanol are usually a result of a situation in which ethanol is made available and the person imbibes it because of curiosity, preconceived notions regarding its effects, and/or events that occur within the context of the situation. The consumption of ethanol produces physiologic sequelae that generate cues that it has been consumed (see below). The physiological event on which we have concentrated is the diffuse arousal and/or behavioral activation that is evident at low doses. We have ascribed the generation of this low-dose response to the activation of certain brainstem NE systems. One can consider that this physiological response can become associated with either pleasurable or aversive consequences of the situation in which the ethanol is consumed. This physiological effect, in and of itself, provides no reinforcement or aversive consequences, and can be considered primarily as a discriminative stimulus. Ethanol consumed at low or moderate doses may also act as a secondary reinforcer in certain situations in which a primary reinforcer is present (Stewart & Grupp,

1981). This secondary reinforcing property of ethanol may again be a consequence of its action on noradrenergic neurons, which would allow for reduced gating of the primary reinforcing stimulus. The activation of the brainstem NE system by low doses of ethanol may produce a generalized arousal resulting in increased perception of reinforcing stimuli.

Continued ingestion during the initial drinking session could, however, allow ethanol levels to rise to the point that ethanol, in certain individuals, would *diminish* the effect of a primary reinforcing stimulus present in those individuals' environment. In the above discussion, we have indicated that certain concentrations of ethanol can diminish release of DA from the DA-containing neurons, but the extent of this effect is dependent on the genetic constitution of the animal. If the genetics of individuals predispose them to this action of ethanol, and if one considers the DA systems of brain to be important in mediating reinforcement, one would expect certain levels of ethanol to diminish reinforcement. At this point in the ethanol dose-response curve, one would also expect aversive pharmacological effects (e.g., disequilibrium) to become evident. The balance of the neurochemical diminution of reinforcement and the sensitivity of the individual to the aversive consequences could be considered as the controlling factor in a decision to continue or terminate drinking. If an individual possesses an inherent resistance to the aversive consequences and/or the physiological capacity to rapidly acquire tolerance during even a single drinking session (i.e., acute tolerance), that individual would be expected to continue to drink until higher levels are attained.

Tolerance to certain of ethanol's actions also develops *across* drinking sessions and, as discussed above, this tolerance development depends both on environment-dependent conditioning and on other cellular adaptive responses to the direct effects of ethanol on the neuronal systems of brain. Although tolerance can easily be demonstrated to the sedative or incoordinating effects, and development of tolerance to actions such as "anxiety reduction" has also been suggested (Lipscomb et al., 1980; Maisto & Scheff, 1977), no tolerance seems to develop to the diffuse arousal or behavioral activating effects (Kianmaa & Tabakoff, 1982; Masur & Boerngen, 1980). Lack of tolerance development is also evidenced in the ability of ethanol to stimulate NE turnover in the brainstem of animals. The increased NE turnover after ingestion is still evident in rats chronically fed ethanol, but the depressant effects of high levels of NE turnover are diminished (Hunt & Majchrowicz, 1974; Pohorecky, 1974). Therefore, the initial physiological cues that ethanol has been ingested remain intact in the chronic drinker, but other physiological consequences are altered with the development of tolerance. This altered physiological mosaic may be a contributing factor to the difficulty with which alcoholics or individuals with "high tolerance" to the incoordinating effects of ethanol discriminate between various blood alcohol concentrations (Lipscomb & Nathan, 1980).

The undiminished discriminative stimulus and the conditioned expectations of "reward" (Kaplan et al., 1984; Maisto et al., 1981) can combine with the developed resistance in the individual to certain aversive and incapacitating actions of ethanol to promote the ingestion of higher doses during a drinking

session. If the level of ethanol reaches the necessary threshold to directly activate the release of DA from the appropriate neuronal systems (see above), one can expect that ethanol will start to act as a primary reinforcer of drinking behavior. Experiencing the primary reinforcing effects would increase the probability of drinking and would reduce controls on the quantity consumed during the drinking episode. The above suggestions resemble the hypothesis proposed by Glatt (1967), that critical blood ethanol levels must be surpassed before physiological factors assume importance in the control of drinking. Below this critical level, situational factors play a more prominent role. Our contention would be that tolerance allows for the attainment of such critical blood ethanol levels. Hence, an understanding of the neurochemical basis of tolerance would have important implications for the understanding of alcoholism.

Certain factors, however, may act to diminish the primary reinforcing effects of high doses. The primary factor, in this regard, would be the development of *tolerance to the activation of DA release* produced by ethanol (see above). Thus, with continued drinking of high doses, a decrease in ethanol's primary reinforcing efficacy can be expected. This development of tolerance to the reinforcing properties of ethanol, coupled with conditioned expectations of its actions, can be visualized as a physiologically derived positive feedback system that promotes the intake of higher and higher amounts. One has to view the multiple factors that initiate and promote ethanol consumption as important targets for intervention in programs of primary prevention and within the context of treatment and secondary prevention programs.

8. IMPLICATIONS FOR TREATMENT

Two treatment goals have been advanced as appropriate end points of alcoholism therapy. The first is abstention and the second is normalization of drinking behavior (i.e., controlled drinking; Pattison, 1976). On the basis of studies demonstrating the slow course of recovery of several physiological and cognitive functions in an alcoholic and the detrimental effects of continued consumption on the process of recovery (Eckardt et al., 1980), the goal of controlled drinking has to be considered with great caution. We will, however, attempt to emphasize aspects of the above-presented hypothesis that impact on both goals.

No matter which of the treatment goals is being pursued, it appears important to extinguish the association between environmental and physiological cues that herald ethanol intake, and the expectation by the individual of impending positive reinforcement. Extinction in classic terms involves the elimination of the positively reinforcing consequences of a behavior. However, we would rather concentrate our concerns on elimination of the cues that generate or initiate the behavior. If the environmental situation and/or the ingestion of low doses of ethanol generate a diffuse physiological arousal,

interpreted as "craving," which an alcoholic uses to make a decision about initiating or continuing drinking (Maisto & Schefft, 1977), one could apply the knowledge of the neuronal systems that mediate the initial arousing effects (e.g., NE) to manipulate the physiological arousal and the subsequent decision about ethanol intake. Pharmacological agents, such as NE synthesis inhibitors or 5-HT uptake inhibitors, may well act by reducing the *initial* cues associated with, or generated by, ethanol intake.

The control of intake, once drinking has begun, has been proposed to rely on occurrence of additional internal physiological changes (cues) that underlie the perception of intoxication (Lipscomb & Nathan, 1980). The perceived intoxication, in turn, influences behavior and the decisions about further drinking. The impaired ability to use internal cues to control intake and level of intoxication has been proposed as an explanation of alcoholic drinking behavior (Lipscomb & Nathan, 1980). We and others (Maisto et al., 1978) suggest that tolerance development may well be responsible for diminished physiological cues regarding intoxication. One treatment approach has been to provide for an alcoholic a means for utilizing external, rather than internal, cues to discriminate blood ethanol levels and control the quantity consumed (Lipscomb & Nathan, 1980). Another treatment approach, which is currently not available, would be to reduce the amount of developed tolerance and to return to the individual the internal cues utilized to control drinking behavior. The approach may be feasible, based on the above-described studies (Lê et al., 1980; Tabakoff & Ritzmann, 1977) of the pharmacological manipulation of ethanol tolerance by 5-HT- and NE-depleting agents and by vasopressin-like peptides (Hoffman et al., 1979).

The other aspect of tolerance development that could impact on treatment success is the acquired ability of the tolerant individual to attain blood ethanol levels that allow ethanol to act as a primary reinforcer and hence generate what has been referred to as "loss of control" drinking. If, as we proposed, this *high-dose effect* of ethanol is mediated through actions on the DA neurons of brain, one may consider use of antidopaminergic drugs to prevent the occurrence of this event. The other approach would, of course, be to prevent the individual from attaining the necessary high blood ethanol levels by increasing the likelihood of aversive consequences. This result is currently accomplished by use of drugs such as disulfiram, but a diminution of tolerance for the aversive consequences would also accomplish the same end.

Finally, we would speculate that those individuals with the capacity to rapidly develop tolerance are poor candidates for controlled drinking treatment programs. These individuals would be the most likely to lose the internal cues that can be used to control intake and would more rapidly develop the ability to attain the levels necessary to generate direct effects on brain reward systems.

In all, the neurobiological hypothesis we have presented attempts to outline a mechanism by which genetically determined physiological events combine with environmental factors to produce positive feedback loops that promote pathological drinking behavior. The major implication of this conceptualization of the etiology of "alcoholism" is that breaking the vicious circle of

drinking, tolerance development, and increased drinking should become a concern in the treatment and prevention of alcohol-related problems.

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3. NEUROBEHAVIORAL THEORY OF ALCOHOLISM ETIOLOGY

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1. PRINCIPAL ELEMENTS OF THE THEORY

Certain behavioral disturbances, if present during childhood and adolescence, increase the risk for alcoholism in adulthood (Alterman & Tarter, 1983; Jones, 1968, 1971; McCord & McCord, 1960; Robins, 1962). These manifest behavioral deficits can be interpreted within a neuropsychological framework and, as will be demonstrated, point to a dysfunction of neural systems lying along the prefrontal-midbrain axis of the brain. Therefore, in the advancing of a comprehensive theory of alcoholism etiology from a neurobehavioral perspective, it is essential that the theory account for a rather diverse array of disturbed psychological capacities, such as the so-called "executive" functions, cognition, motivation, and emotion, as well as arousal processes.

The neurobehavioral disturbances comprising the vulnerability to alcoholism are hypothesized to be inherited. These behavioral disturbances, reflecting the final link in the chain of genetically determined biological processes, consist of lability, impersistence, disinhibition, hyperactivity, and attentional deficits, in addition to cognitive impairments similar to those found in persons with lesions in the anterior-basal brain region. At the neurochemical level of biological organization, the genetic predisposition is conjectured to involve a disequilibrium of neurotransmitter mechanisms, and at the physiological level of analysis, the susceptibility is postulated to be manifested as a disruption of homeostatic mechanisms responsible for regulating arousal. Thus, although the focus of the present discussion is on neuropsychological

parameters, the point needs to be underscored that the theory strives for comprehensiveness and, as such, attempts to relate behavioral variables to neurochemical and neurophysiological processes, as well as to the underlying neuroanatomical substrate. Hence, the genetic vulnerability to alcoholism is conceptualized herein as a multi-process disturbance that is expressed across different levels of biological organization, and that culminates in disturbed psychological functioning.

Before we present the specific details of the neurobehavioral theory of alcoholism vulnerability and examine the evidence regarding the underlying biological mechanisms, it is important to first acquaint the reader with the conceptual framework and methodology of neuropsychological investigation.

1.1 DEFINITION, METHODS, AND SCOPE OF NEUROPSYCHOLOGY

Neuropsychology is a hybrid discipline, historically evolving from efforts to interrelate brain (neurological) and behavioral (psychological) processes. A major objective of neuropsychological research is to determine how psychological processes are organized into functional systems in the brain. Hence, in its broadest context, neuropsychology is defined as the study of brain-behavior relationships.

An extensive literature pertaining to how the brain is functionally organized has been accumulated during the past century of clinical and experimental research. Based on this body of knowledge, psychological tests and batteries have been developed that are capable of reliably and validly lateralizing and localizing cerebral lesions (Berent, 1981; Lezak, 1983; Luria, 1966). With an understanding of brain functional organization, it is thus possible, from the administration of a battery of tests, to interpret a pattern of performance deficits such that a specific dysfunctional or lesioned neuroanatomical site or system can be identified. This method of clinical diagnosis is successful at an order of accuracy that is superior to the EEG, the angiogram, and the neurological examination (Filskov & Goldstein, 1974). In conditions involving either biochemically or metabolically mediated brain dysfunction, and early or subtle forms of brain atrophy, neuropsychological measures are additionally superior to computerized tomography (CT) scans, which illustrate only the morphological characteristics of the brain.

Neuropsychological test measures have, for quite some time, been routinely utilized to assess for the presence, and to determine the location, of cerebral lesions in neurological patients. They have recently also been successfully employed to elucidate the nature of cerebral dysfunction in the so-called "functional" disorders, such as obsessive-compulsive syndrome (Flor-Henry et al., 1979), schizophrenia (Asarnow, 1983; Holzman & Tarter, 1982), depression (Schweitzer, 1979), hysteria (Flor-Henry et al., 1981), and sociopathy (Yeudall, 1980; Yeudall et al., 1982). Although these latter psychopathological conditions are usually not associated with observable structural brain lesions, neuropsychological measures nonetheless have been able to reveal cognitive impairments that are, by their manifest pattern, suggestive of cerebral dysfunction. The point to be underscored is that neuropsychological measures not only are applicable to situations where there is suspected morphological damage,

but are valuable as well for clarifying the behavioral and cognitive sequelae of neurochemical and physiological disturbances.

Neuropsychological research in the field of alcoholism has, in the past, been almost entirely concerned with the *consequences* of chronic drinking. In this regard, much has been learned about the origins, pattern, and severity of the cognitive deficits associated with alcoholism (Tarter & Ryan, 1983; Wilkinson & Carlen, 1980a,b), the neuropsychological subtypes of alcoholics (Lishman, 1981; Wilkinson & Carlen, 1980b), the relationship between the pattern and degree of behavioral impairment and neuroradiological indices of atrophy (Ron, 1983; Wilkinson & Carlen, 1980c), and the association between certain biochemical indices associated with liver dysfunction and nutritional states and test performance (Albert et al., 1982; Gilberstadt et al., 1980). The question, however, of whether there are neuropsychological deficits that *antedate* drinking onset has not been empirically addressed in a systematic fashion. Consequently, in a proposed neuropsychological theory of alcoholism etiology, the current state of knowledge dictates that evidence be interwoven from diverse sources incorporating investigations that were not originally intended to examine neurobehavioral relationships. Although the paucity of direct neurobehavioral research limits the conclusions that can be drawn, it will nonetheless be seen that the neuropsychological theory advanced herein not only is heuristic, but also is capable of integrating the available behavioral findings with the biological processes that have been implicated to predispose to alcoholism. Thus, the proposed neuropsychological theory can be said to have a high degree of explanatory power.

1.2 IMPLICATIONS OF A NEUROPSYCHOLOGICAL PERSPECTIVE OF ALCOHOLISM ETIOLOGY

There are a number of ramifications of a neuropsychological theory of alcoholism etiology. First, any overt behavioral deficit may be the product of a disturbance at one or more levels of biological organization. Structural, neurochemical (neurotransmitter and endocrine), or electrophysiological disturbances may thus underlie impaired neuropsychological functioning. Second, by implicating neuropsychological disturbances prior to the onset of alcohol consumption, the theory calls into question the validity and generalizability of the numerous deficits that have been found in alcoholics and in social drinkers (MacVane et al., 1982; Parker & Noble, 1977; Parker et al., 1980; Ron, 1977; Ryan & Butters, 1983; Tarter, 1975; Tarter & Alterman, 1984; Wilkinson & Carlen, 1980a,b). Although there is no doubt that the chronic and excessive consumption of alcohol exerts a deleterious effect on neuropsychological capacities, the issue of whether *all* of the reported impairments are a consequence of the alcoholism has only recently been addressed (Tarter & Alterman, 1984).

A third ramification concerns the breadth of behavioral processes covered by neuropsychological measures. The neuropsychological assessment has traditionally focused on only cognitive capacities, although recent brain-behavior research has indicated that there is also cerebral specialization of emotional and motivational processes (Campbell, 1982; Gruzelier & Flor-

Henry, 1979). Hence, for disorders in which affective-motivational processes are involved as etiological determinants, it is essential, therefore, to broaden the perspective of neuropsychology to include these latter processes in striving for a comprehensive explanatory framework. And fourth, neuropsychological investigation has, until recently, confined itself almost completely to acquired brain dysfunctions due to disease processes originating during intrauterine development or postnatal life. However, brain maturation is marked by both inter- and intra-individual variability that presumably reflects, to a large extent, genetic factors. In addition, cognitive (De Fries et al., 1981; Diamond et al., 1983; Horn, 1982; R. Wilson, 1981, 1983) and personality (Dixon & Johnson, 1980; Eaves et al., 1978; Eysenck, 1983) processes are influenced to a substantial extent by genetics. In view of the strong evidence for an inherited predisposition to alcoholism (Cloninger et al., 1981; Goodwin, 1983; Grove & Cadoret, 1983), it is therefore reasonable to assume that the susceptibility, discussed herein with respect to the gene-behavioral pathway, is explicable in terms of an innately determined rate and pattern of brain maturation. Hence, neurobehavioral research in general, and the specific theory described in this discussion, forms a natural link between genetics, the neuroanatomical substrate, intervening biological mechanisms, and the developmental acquisition of diverse psychological processes encompassing cognition, emotion, motivation, and organismic arousal regulation.

2. HISTORICAL DEVELOPMENT OF THE THEORY

The notion that neuropsychological factors play an etiological role in alcoholism is rather novel. Indeed, this discussion is the first attempt to organize the existing literature within a neuropsychological framework. As mentioned previously, neuropsychological investigations in the past have been confined almost entirely to the consequences of alcoholism. An examination of the genetic, psychological, and physiological research indicates, however, that there is reason to suspect that neurobehavioral disturbances are associated with alcoholism etiology. Below are summarized a number of findings that implicate central nervous system dysfunction prior to the commencement of drinking.

1. Many alcoholics were hyperactive as children (Goodwin et al., 1975; Tarter et al., 1977; Wood et al., 1976). Also, a hyperactive child is more likely than a nonhyperactive child to have an alcoholic biological father, even if the child was adopted away from the parent at a very early age (Cantwell, 1972; Morrison & Stewart, 1973). Hyperactive adolescents are also more likely to abuse alcohol than their peers (Blowin et al., 1978; Mendelson et al., 1971).
2. Children of alcoholics, who themselves are at heightened risk to develop this disorder, exhibit faster waveform activity on the EEG (Gabrielli et al., 1982).
3. Nonalcoholic relatives of alcoholics, tested while sober, exhibit greater static ataxia than relatives of nonalcoholics (Lipscomb et al., 1979).

4. Patients with essential tremor have a higher incidence of alcoholism, as well as alcoholism in their first-degree relatives, than individuals without essential tremor (Schroeder & Nasrallah, 1982). This condition also frequently antedates problematic drinking in alcoholics (Nasrallah et al., 1982).
5. Young left-handers are more prone to alcohol abuse than right-handers (Lee-Feldstein & Harburg, 1982). There is also a higher than expected proportion of left-handed alcoholics (Bakan, 1973).
6. Adolescent sons of alcoholic fathers are more neuropsychologically impaired than offspring of nonalcoholic fathers (Tarter et al., 1984), and also demonstrate lower levels of academic achievement (Hegedus et al., 1984).
7. Approximately one out of three alcoholics meets DSM-III diagnostic criteria for an attention deficit disorder, residual type (Wood et al., 1983). Attentional disturbances in childhood are also related to the risk for alcoholism (Wender et al., 1981; Wood et al., 1976).
8. Nonalcoholic first-degree relatives of alcoholics perform more poorly on neuropsychological tests measuring abstracting and problem solving and perceptual-motor capacity than do nonalcoholics who do not have a family history of alcoholism (Schaeffer et al., 1984). This finding indicates that neuropsychological deficits are more likely to be evident in adults who are at risk for developing alcoholism.
9. Prealcoholics often present a behavioral disposition of impulsivity, disinhibition, high activity, emotional lability, and aggressiveness (Berry, 1967; Jones, 1968; McCord & McCord, 1960; Ricks & Berry, 1970; Robins et al., 1962; Vaillant, 1983); these characteristics, though not necessarily implicative of a CNS dysfunction, are at least congruent with such a hypothesis.
10. Antisocial and psychopathic propensities frequently antedate the commencement of problematic drinking (Cloninger et al., 1981; Penick et al., 1983; Robins et al., 1962), disturbances that themselves have been related to neuropsychological impairment (Gruzelier & Flor-Henry, 1979).

In addition to the above findings, the possibility that neuropsychological deficits predate drinking is further suggested by the fact that no substantial or consistent association has been found between alcohol consumption variables (e.g., chronicity, quantity) and test performance (Eckardt et al., 1978). The above considerations support the contention that there may be a central nervous system disturbance prior to the onset of alcoholism for some individuals. In §5, Research Results, the above observations will be systematized and expanded on, and supplemented with additional psychological and physiological evidence, to demonstrate how the pattern of manifest deficits is congruent with the theory of an anterior-basal brain dysfunction. Suffice it to say at this juncture, efforts aimed at elucidating alcoholism etiology from a neuropsychological perspective did not evolve from a program of planned research, or from a particular theoretical position, but instead were inspired by an inspection and appraisal of existing data and how they could be most parsimoniously interpreted.

3. CHARACTERISTIC RESEARCH METHODS

A neuropsychological theory of alcoholism etiology assumes that: (1) the manifest behavioral disturbances are prodromal to alcoholism; and (2) the behavioral deficits are interpretable from the standpoint of a dysfunction of one or more neural systems. This section will review the paradigms that have been employed and the behavioral characteristics that appear to predispose to alcoholism. Next, the techniques of neuropsychological research will be described. Since no investigations have been conducted that specifically attempted to prospectively link neurobehavioral processes in childhood to alcoholism in adulthood, the conclusions drawn herein are necessarily limited by the application of post hoc interpretations. Thus, rather than being a definitive explanatory exposition, the proposed theory should be evaluated at this stage for its integrative and heuristic value in guiding future systematic investigation.

3.1 LONGITUDINAL INVESTIGATION

The longitudinal study is the most powerful paradigm for demonstrating an association between childhood behavior characteristics and psychiatric outcome. Unfortunately, only a few investigations utilizing this paradigm have been conducted with respect to alcoholism (Jones, 1968; Loper et al., 1973; McCord & McCord, 1960; Vaillant, 1983; Wood et al., 1983), and of these few studies, only one has followed a cohort of subjects to the age of maximum lifetime risk (Vaillant, 1983). The finding that hyperactivity (Jones, 1968), attentional disturbances (Wood et al., 1983), impulsivity (McCord & McCord, 1960), and behavioral disinhibition (Loper et al., 1973) in childhood and adolescence predate alcoholism suggests that there may be an underlying central nervous system dysfunction in prealcoholics. Although caution must be exercised regarding the interpretation of the above behaviors in terms of neurological mechanisms in the absence of direct measurement, it is nonetheless one viable explanation that, as will be seen, gains validity when considered in the context of other converging findings. In addition, the demonstration of an association between delinquency in adolescence and alcoholism in adulthood (McCord & McCord, 1960; Robins et al., 1962) can be interpreted as suggestive of a predisposing CNS dysfunction, especially when considered in the context of studies reporting neuropsychological deficits (Krynicki, 1978; Spellacy, 1977) and a high incidence of learning disability (Robins, 1966; Yule & Rutter, 1968) in the antisocial disorders.

3.2 GENETIC PARADIGMS

Twin (Jonsson & Nilsson, 1968; Kaij, 1960; Partanen et al., 1966), adoption (Bohman, 1978; Cadoret & Gath, 1978; Cadoret et al., 1980; Goodwin et al., 1973, 1974; Schuckit et al., 1972), and familial (Amark, 1951; Bleuler, 1955; Pitts & Winokur, 1966) studies all point to a genetic predisposition to alcoholism. However, there have, as yet, been no formal neuropsychological investigations employing genetic paradigms. Evidence reviewed by Alterman and Tarter (1983) nonetheless indicates that the transmission of the

vulnerability to alcoholism may be expressed in the form of a number of behavioral disturbances. These behavioral disturbances can also be accommodated within the known dimensions of temperament (Tarter et al., 1985), a point illustrating the explanatory potential of a behavioral-genetic research strategy. Furthermore, the overtly expressed temperament traits, consisting of heightened activity level, high emotionality, low soothability, low attention span and persistence, and low sociability, are interpretable from a neurobehavioral perspective and, according to Tarter et al. (1985), implicate the anterior-basal brain system. The above-described behaviors, all being of largely genetic origin, appear to comprise noncognitive aspects of the diathesis to alcoholism, subserved by limbic, diencephalic, and midbrain mechanisms.

There is suggestive evidence that certain cognitive deficits may, to some extent, be inherited as well (De Fries et al., 1976, 1978). Whether or not cognitive impairments directly render the person more susceptible to becoming alcoholic as, for example, by limiting the development of more adaptive problem-solving strategies, or for any other reason, is at present unknown. It may be that inherited neuropsychological deficits, if indeed found to antedate alcoholism, are merely interesting epiphenomena, and hence not directly related to the risk for a negative outcome in adulthood.

Because there is parental alcoholism in up to 50% of alcoholics, it is possible that physical abuse and head trauma due to a chaotic family environment may also contribute to the reduced cognitive capacity in the proband. Another possible nongenetic factor pertains to neurological damage concomitant to a partial fetal alcohol syndrome consequential to being born to an alcoholic mother who drank during gestation. Also, alcoholics are often delinquent or antisocial as adolescents—a disposition that exposes them to neurological injury and cognitive impairment resulting from a violent and deviant lifestyle. Thus, in order to determine if there are innate cognitive disturbances or a particular cognitive style that antedates alcoholism, it is imperative that genetic paradigms be employed so that the contribution of nongenetic factors also can be ascertained.

3.3 HIGH-RISK PARADIGM

This research strategy selects individuals for study who, on the basis of either a priori assumption or empirical evidence, are considered to be at increased risk to develop the disorder under inquiry. Alcoholism, being to a large extent a familial disorder (Cotton, 1979), can, for example, be studied by measuring neuropsychological capacity in the nonaffected relatives of alcoholics, such as siblings, parents, or children. This approach can yield valuable information regarding the neurobehavioral characteristics associated with the risk for alcoholism. The potential value of this strategy of research is exemplified by the findings of Schaeffer et al. (1984), who demonstrated that nonalcoholic adult relatives of alcoholics perform more poorly on certain neuropsychological measures than nonalcoholic relatives of nonalcoholics. Another example of the value of this paradigm is illustrated by Lipscomb et al. (1979), who observed greater static ataxia in nonalcoholic young adult relatives of alcoholics than in relatives of nonalcoholics. A higher incidence of essential

tremor has also been observed in the relatives of alcoholics (Schroeder & Nasrallah, 1982). These latter studies aptly illustrate that individuals who presumably possess a high genetic loading, but for whatever reason did not develop alcoholism, exhibit evidence for a CNS dysfunction.

In studying children who are at elevated risk, it is not possible, unless the study is integrated into a longitudinal paradigm, to ascertain if the manifest neuropsychological characteristics are associated with an outcome of alcoholism. For instance, adolescent male offspring of alcoholic fathers have been found to have more neuropsychological impairments (Tarter et al., 1984), and to perform less well on an academic achievement test (Hegedus et al., 1985). Unless these adolescents are tracked longitudinally, it is not possible to determine if the manifest impairments augment the risk for alcoholism.

One variant of the high-risk paradigm, which makes no assumptions about genetic loading, involves selecting subjects according to particular biological or psychological characteristics. The particular criteria utilized are based on evidence that the presence of the putative characteristics are associated with a high risk for alcoholism. Taking this approach, Sher and Levenson (1982) composed two groups of nonalcoholic young adults according to their MacAndrew Alcoholism Scale score from the MMPI and the Socialization Scale score from the California Psychological Inventory. An elevated score on the MacAndrew scale has been reported to be associated with a high risk for alcoholism (Hoffmann et al., 1974), as has low socialization (McCord & McCord, 1960). Upon examination for a differential psychological and physiological reaction to alcohol, it was found that there were distinguishing features between presumed high- and low-risk subjects in terms of alcohol's subjective and reinforcing effects.

An advantage of the high-risk paradigm is that it affords the opportunity to investigate predisposing risk factors across multiple levels of biological organization. Hence, neurological (Lipscomb et al., 1979; Schroeder & Nasrallah, 1982), metabolic (Schuckit & Rayses, 1979), biochemical (Schuckit et al., 1982), and electrophysiological (Gabrielli et al., 1982) studies of relatives of alcoholics have been conducted demonstrating that persons susceptible to developing alcoholism can be distinguished from their peers. In addition, the high-risk paradigm readily lends itself to expeditious data collection, inasmuch as it is not encumbered by the numerous conceptual and empirical limitations inherent in the twin and adoption paradigms.

3.4 NEUROPSYCHOLOGICAL METHODS OF RESEARCH

The paradigms described above are useful for the identification and tracking of subjects who are genetically predisposed to alcoholism. In order to test the theory that neuropsychological factors are involved in the etiology of alcoholism, it is necessary to utilize neurobehavioral measurement techniques in the context of genetic and longitudinal paradigms. Although the neurological mechanisms responsible for the manifest behavioral impairments cannot be determined from these procedures, neuropsychological techniques can accurately localize the brain system and region that is disrupted (Lezak, 1983).

The capacity to lateralize and localize cerebral pathology is the culmina-

tion of over a century of research on brain-behavior relationships. Much of what is known of the behavioral sequelae of the various brain disorders has been learned from the study of children and adults suffering from focal and diffuse neuropathology. Electrical stimulation of the brain, as well as the elucidation of the behavioral effects of surgical ablation of brain tissue from neurological and psychiatric patients, has also substantially added to our understanding of how the brain is functionally organized. Controlled information input, utilizing techniques such as tachistoscopic and dichotic stimulus presentation, has further increased our understanding of neuropsychological organization. In addition, the behavioral effects of electrical and chemical brain stimulation, as well as experimentally induced lesions in animals, have enhanced our knowledge of brain-behavior relationships. Against this background of literally thousands of neurological investigations, there has emerged a body of knowledge delineating the relationship between neurological pathology and behavior impairment. On the basis of this understanding of brain-behavior relationships, it is consequently possible to utilize behavioral measurement to localize the neuroanatomical substrate that is pathological. Indeed, certain standardized neuropsychological batteries, such as the Halstead-Reitan Battery (Reitan, 1955) and Luria-Nebraska Battery (Golden, 1980), as well as numerous other specific tests (Lezak, 1983), have proven to be both valid and reliable in this regard.

By administering a battery of neuropsychological tests, it is thus possible to localize and lateralize a cerebral lesion or a dysfunctional brain system. In addition, an understanding of functional neuroanatomical systems enables the examiner to select test measures that are capable of testing a particular brain dysfunction hypothesis. For example, if persons at high risk for becoming alcoholic are hypothesized to have an anterior-basal brain dysfunction, then such persons would be expected to exhibit certain cognitive impairments, as well as demonstrate difficulties in planning, implementing behavior programs, and self-regulation — the so-called “executive” functions (Lezak, 1982). By systematic selection of the appropriate measurement instruments, it is therefore possible to determine if a dysfunctional brain system is associated with alcoholism vulnerability.

In summary, neuropsychological techniques can reveal the locus, but cannot independently determine the cause, of a dysfunctional or pathological neuroanatomical substrate. By employing neuropsychological procedures in conjunction with genetic, high-risk, and longitudinal paradigms, it is possible, however, to ascertain if there are neurobehavioral correlates of the risk for developing alcoholism.

4. BOUNDARIES OF THE THEORY

The proposed neuropsychological theory of alcoholism vulnerability applies to only a subset of individuals in the population. Because alcoholism is a very heterogeneous disorder, varying greatly between individuals in terms of

purported etiology and clinical characteristics, it is unreasonable to expect that any one theory could be both comprehensive and, at the same time, specific enough to define the nature of the vulnerability for all alcoholic individuals. Consequently, the theory advanced herein pertains to individuals who meet the following criteria: an early-age onset of heavy drinking without an identifiable precipitating cause; severe manifestations of alcoholism symptomatology; and antisocial propensities. The proposed theory does not claim to apply to cases of alcoholism that are secondary to another psychopathological condition, or where dependence arose from habit or in response to stress.

A neuropsychological theory addresses only organicistic characteristics. Hence, it does not claim to account for all aspects of alcoholism etiology. For alcoholism to develop, alcoholic beverages must be reasonably accessible in society. Facilitative influences, such as ambivalence about drinking by family and peers as well as by the socioeconomic and cultural macrosystems, also affect drinking behavior and ultimately the risk for becoming an alcoholic. Thus, alcoholism can be viewed as the end point in a chain of events involving the interaction between numerous environmental factors and a genetically vulnerable organism. Conceptualizing alcoholism etiology in this manner (e.g., from a diathesis-stress perspective) affords the opportunity to investigate etiology as a multifactorial phenomenon, and to quantify the interaction and impact of the plethora of variables that could either exacerbate or attenuate the risk. Neuropsychological impairment, considered in this context, is thus but one of a number of etiologically relevant factors.

The proposed neuropsychological theory encompasses the domain of psychobiological processes and capacities mediated by the neural systems coursing along the prefrontal-midbrain axis. As such, the theory accommodates a variety of behavioral phenomena that, though superficially disparate (cognitive, affect, arousal, etc.), are united by the fact that they are functionally integrated within this neuroanatomical system.

The theory that an anterior-basal dysfunction underlies the vulnerability to alcoholism has at least three major research ramifications. First, it raises the possibility that the dysfunction, hypothesized to be neurochemical in nature, renders the person highly susceptible to the adverse effects of alcohol consumption. In one preliminary investigation, Begleiter, Porjesz, and Kissin (1982) found that familial alcoholics (those presumably but not necessarily possessing a greater genetic loading for this disorder) exhibited more brain atrophy than nonfamilial alcoholics, even when age and duration of drinking history were controlled. It is also interesting to note that Wilkinson and Carlen (1980a,b) have identified two neuropsychological subtypes, these being a premature aging profile and an amnesic profile. The fact that the brain atrophy appeared to occur relatively early in the drinking career in the amnesic type points to a particular vulnerability to alcohol's deleterious effects in these individuals. Unfortunately, no information about family history or genetic predisposition was obtained. However, both of these investigations indicate that there may be a susceptibility in some alcoholics to suffer cerebral atrophy, a vulnerability that could stem from a neurochemical dysfunction that is hypothesized to exist before the onset of alcohol consumption.

A second and somewhat related ramification is that alcoholic individuals possessing the anterior-basal dysfunction are more susceptible than other persons to the acute physiological effects of alcohol. It is conjectured that such alcoholics are more inclined than other alcoholics to experience symptoms such as blackouts, delirium tremens, and withdrawal — disturbances that appear to be the consequence of a disruption of limbic and diencephalic mechanisms (Segal et al., 1970). The finding that these symptoms of alcoholism are not highly correlated with duration of alcoholism and are very often found in young alcoholics (Bergman & Agren, 1974) suggests that there may be a differential vulnerability to these symptoms in some alcoholics.

Third, a dysfunctional anterior-basal system may result in a differentially greater reinforcing response to the acute effects of alcohol, since the brain mechanisms subserving reward are contained within this neural system. Hence, it is speculated that the reinforcement consequences of alcohol consumption, and the associated emotional and motivational changes from intoxication, are different for persons at high risk for alcoholism in comparison with the rest of the population. Evidence presented by Sher and Levenson (1982) tends to confirm this association between alcoholism vulnerability and alcohol's reinforcing effects. The finding by Tarter et al. (1977) of a euphoric response to alcohol in primary alcoholics also suggests that drinking may be particularly reinforcing for some individuals.

Although the neuropsychological theory of an anterior-basal dysfunction encompasses a broad range of cognitive, motivational, and emotional variables, and is specific to a particular type of alcoholic — namely, the early-onset antisocial alcoholic — the point needs to be underscored that it is not, in a strict sense, an etiological theory. Rather, what is proposed is that this central nervous system dysfunction constitutes only the *vulnerability* or predisposition to develop alcoholism. Hence, the theory, as proposed, is more concordant with a probabilistic diathesis-stress model of psychopathology than an invariant cause-and-effect model. In addition, there is the possibility, as yet untested, that individuals possessing the susceptibility are more deleteriously affected by chronic alcohol consumption, and also respond differentially to the acute effects of ethanol. Although admittedly somewhat speculative, the theory proposed raises the possibility that the CNS vulnerability not only influences the propensity to initiate problematic drinking but also augments the potential to suffer greater adverse neurological consequences from drinking.

Because of the impossibility of direct manipulation of biological variables and direct measurement of biological processes in human neuropsychology research, the need to develop an animal model is paramount. This model can be achieved either by investigating different strains of animals to determine if alcohol preference is correlated with the behaviors theorized to constitute the diathesis for alcoholism or, alternatively, through selective breeding. The former research program, for example, would be directed to determining if animals that are rendered highly active, excitable, and slow to learn an inhibitory response (e.g., by chemical or surgical lesions) undergo a change in alcohol preference. If the behavioral features associated with the vulnerability to alcoholism in humans are the same as the behaviors related to alcohol

preference in animals, that would suggest that similar neural structures may be involved. For example, Gorenstein and Newman (1980) and Newman et al. (1983) have proposed a very elaborate theory of human psychopathology based upon the notion of intrinsic neural disinhibition of a system comprising the prefrontal cortex, hippocampus, and medial-septum. Employing an animal model, they observed that rats with septal lesions behaved similarly in certain key respects to psychopathic individuals. Behavior in the lesioned animals was primarily determined by the most prominent motivationally significant cue, and the result was an inability of the animals to delay gratification. The septal area comprises an integral component of the frontal-limbic system, and a dysfunction of this system has been implicated to underlie psychopathy (Hare, 1970) and hyperactivity (Rosenthal & Allen, 1978). Inasmuch as these latter disorders are associated with risk for alcoholism, it is readily seen how experimentation with animals can elucidate the neurobehavioral correlates of the predisposition.

Selective breeding is another research strategy that could help determine the behavioral characteristics and genotype associated with the susceptibility to alcohol dependence. This strategy involves clarifying the neurochemical and physiological characteristics of animals that exhibit a predilection to voluntary alcohol consumption. Thus, even though human neuropsychological research cannot directly measure the underlying biological mechanisms, it nonetheless could facilitate the search for such mechanisms through the development of an animal model.

5. RESEARCH RESULTS

Prior to a review of the research evidence, it is necessary to first consider the question of whether the anterior-basal brain region, consisting of a substantial proportion of cerebral mass, is a functionally integrated system. Two lines of evidence suggest that this is indeed the case. First, it has been convincingly argued that the prefrontal region of the cortex is the association area for the limbic system (Fulton, 1952; Pribram, 1960). Consequently, it is not surprising that limbic and prefrontal lesions produce similar behavioral changes (Pribram, 1960, 1969). Second, tissue degenerative techniques have revealed the organization of the fibre tracts that emanate from the prefrontal cortex. Fibres from the dorsolateral area project to the superior temporal gyrus; those from the orbital area project to the middle and inferior temporal gyri; both tracts project to the temporal lobe via the fasciculus uncinatus (Nauta, 1962).

A frontal-hypothalamic tract has also been demonstrated (Le Gros Clark, 1948). In fact, it has been hypothesized by Le Gros Clark (1948) that the hypothalamus functions as a primary projection area, just as does the striate cortex for the visual system. Nauta (1962) supports this thesis with the following statement:

It will suffice here to point out that the hypothalamic neurons in question

combine characteristics of nerve cells and secretory cells and are thought to elaborate humoral principles, some of which are hormones, or their precursors, in their own right, whereas others serve as transmitter substances affecting the secretory mechanisms of the anterior pituitary. Whatever their glandular characteristics, these cells must, nevertheless, be regarded as the last neuronal link in the major endocrine motor organization and hence, as an unusual sort of "final common path." Experimental as well as clinical data suggest that although obviously the whole of the neocortex is engaged in the overall function of differential anticipatory behavior, rostral parts of the frontal, and to some extent also the temporal lobes are of some special significance for the "anticipation and planning (MacLean, 1958) as it applies to both the self and the species." It is, therefore, of great interest that the only known neocortical projections to the "limbic system-midbrain circuit," in which the hypothalamus lies imbedded, arise from precisely these cortical areas. (p. 207)

A hypothalamic-frontal tract has been identified (Le Gros Clark & Meyer, 1950), synapsing first in the dorsomedial nucleus of the thalamus before projecting to the prefrontal areas. Three important tracts emanating from the dorsomedial nucleus of the thalamus also project to the prefrontal regions: the *pars paramellaris* to the frontal eyefields, the *pars parvocellularis* to the dorsolateral region, and the *pars magnocellularis* to the orbital region. It is also of particular interest that the frontal eyefields and midbrain structures, particularly the reticular activating system and superior colliculus, are connected and together subserve and integrate such complex functions as arousal, attention, visual search, and spatial analysis (Crowne, 1983).

Thus, there are specific reciprocally innervated connections between prefrontal, diencephalic, and midbrain structures. From a functional standpoint, it is apparent that this system is integrally involved in mediating cognitive processes via the neocortex, emotional and motivational processes via the prefrontal and limbic regions, and organismic arousal via the diencephalon and the midbrain. Therefore, in advancing this substrate as being dysfunctional in individuals vulnerable to alcoholism, it is essential that a neuropsychological analysis embraces the above diverse aspects of psychological functioning.

The following review of the evidence implicating the anterior-basal system as the underlying substrate is organized into three sections: (1) prefrontal dysfunction; (2) limbic and diencephalic dysfunction; and (3) midbrain dysfunction. This division is made for convenience and clarity of exposition only, since these brain regions, as emphasized above, actually comprise an integrated functional system.

5.1 NEUROBEHAVIORAL CORRELATES OF THE PREFRONTAL REGION

Over a century ago, Sir Hughlings Jackson postulated that the anterior brain region exerted a regulatory role over behavior by modulating the activity of lower brain structures. Through primarily inhibitory mechanisms innervating diencephalic, midbrain, and spinal structures, the prefrontal cortex was hypothesized to mediate choice behavior and self-regulation (Jackson, 1958). Extensive research conducted on both humans and animals has since confirmed Jackson's position. In the syntax of contemporary neuropsychology, the

prefrontal region subserves the "executive" functions of the brain: that is, planning or goal formulation, initiating and sustaining behavior programs, and self-monitoring and self-evaluation of behavior (Lezak, 1982). Luria (1966) emphasizes the importance of language mediation as being integral to behavioral self-regulation and goal persistence. Of particular interest is Luria's assertion that the maturation of the anterior frontal brain region does not take place until the fifth or last stage of development, this being the time of early adolescence. This latter point has especially salient ramifications for the present theory, since *it suggests that the cerebral dysfunction evidenced behaviorally in high-risk individuals would first be detected in adolescence, at which time age-appropriate capacities have not been acquired.*

In light of the above, it is interesting to note that the neuropsychological impairments observed by Schaeffer et al. (1984) in nonalcoholics who had a family history of alcoholism were found in the "higher" cognitive functions, and were primarily circumscribed to a cluster of tests that are sensitive to anterior brain dysfunction. These tests included the Category Test of the Halstead-Reitan Battery, the Conceptual Level Analogy Test, the Abstraction Test of the Shipley Institute of Living Scale, Levine's Hypothesis Test task, the WAIS Block Design, and a word finding test. It was further observed that there was no significant interaction between the effects of alcoholism in the subject and the familiality variable, which finding suggests that the neuropsychological deficits are independently and additively determined by these two factors.

Pathology or dysfunction of the anterior frontal brain region results in behavioral and cognitive disturbances that are similar to those found in persons at risk for alcoholism. One frequent sequela of prefrontal pathology is hyperactivity. Hyperactive adolescents are more likely to abuse alcohol than other teenagers (Blowin et al., 1978; Mendelson et al., 1971). Childhood hyperactivity also appears to be a risk factor for the development of alcoholism (Goodwin et al., 1975; Jones, 1971). In addition, hyperactive male children have a relatively high incidence of alcoholism in their biological fathers, but not their adoptive fathers (Cantwell, 1972; Morrison & Stewart, 1973). Although the etiological mechanisms underlying hyperactivity are not completely understood, it appears to be associated with a disruption of arousal mechanisms (Douglas, 1980) and a deficiency in rule-governed behavior (Barkley, 1981). On the basis of the above findings it is therefore reasonable to conjecture that hyperactivity, implicated as one of the risk factors for alcoholism, may involve an anterior brain dysfunction.

Evidence obtained from two other lines of research also points to a dysfunction in the anterior brain region. First, the pattern of neuropsychological deficits found in psychopathy — a disorder that is often comitant with alcoholism, predates alcoholism, and is frequently present in the families of alcoholics — implicates an anterior brain dysfunction (Yeudall, 1980). And second, two recent neuropsychological studies of delinquent offspring of alcoholic fathers have revealed impairments consistent with an interpretation of an anterior dysfunction (Tarter et al., 1984; Hegedus et al., 1984). In these latter studies, the sons of alcoholics and of nonalcoholics were very similar on WISC or WAIS measures of intelligence. However, the former subjects

performed significantly more poorly on several subtests of the Peabody Individual Achievement Test (PIAT), a standardized measure of educational achievement. The low performance of the alcoholics' sons on the PIAT could not be explained on the basis of a disrupted family environment, psychopathology, or behavioral disturbance. Deficits were also detected on tests of attention, short-term memory, and language expression. While these latter results should be viewed as preliminary, they are intriguing inasmuch as they provide the first direct evidence for the presence of neuropsychological impairments in individuals at heightened risk to become alcoholic.

Behavioral disinhibition, restlessness, attentional disturbances, and impulsivity have been reported in prealcoholics (Jones, 1968; Loper et al., 1973; McCord & McCord, 1960) as well as in persons who are at elevated risk for alcoholism (Goodwin et al., 1975). That these latter behavioral disturbances are often sequelae of anterior brain pathology raises the possibility that the same neuroanatomical system is disrupted in individuals vulnerable to alcoholism.

Studies of alcoholics also implicate a frontal pathology. These investigations must, however, be interpreted cautiously, since it is not possible to completely separate the consequences of alcohol abuse on test performance from impairments that may have antedated problematic drinking. Nonetheless, it has frequently been demonstrated that the performance of alcoholics on several neuropsychological measures sensitive to anterior brain pathology is unrelated, or only poorly related, to chronicity or other drinking history variables, and this evidence lends plausibility to the hypothesis that the impairments are not entirely the consequence of longstanding drinking excess.

Tests of cognitive style, such as the rod and frame test (RFT), reliably discriminate alcoholics from nonalcoholics (Sugerman & Schneider, 1976). On the RFT, the person must align a rod to its true vertical position, despite a distracting background frame that is in varying degrees of tilt. The demonstration that neither the duration of drinking history (Karp & Konstadt, 1976), nor the duration of sobriety (Karp et al., 1965a), nor the acute administration of alcohol (Karp et al., 1965b) is associated with performance on the RFT suggests that the poor performance is a stable characteristic of alcoholics, and thus possibly may have preceded the onset of alcoholism (Witkin et al., 1959). In addition, competent performance on this task, what with its high demands for attention and spatial analysis, requires the integrity of the frontal eyefields, which, as noted earlier, are connected via the *pars paramellaris* to the diencephalon, which is hypothesized to be dysfunctional in persons at risk for alcoholism.

It is interesting to note that the performance of alcoholics on the RFT is not immutable to change. Jacobson et al. (1970) found that if alcoholics were allowed a 1-hour period of sensory deprivation, during which time they were instructed to focus on interoceptive cues, their performance significantly improved upon retesting. It has also been shown that RFT performance improves if alcoholics are given an "internal orientation" instructional set (Reinking, 1977). The results of the latter two studies suggest that alcoholics do not ordinarily focus attention inwardly in monitoring their perceptions. In

addition, the demonstration that alcoholics are impaired at estimating the volume of consumed nonalcoholic beverages (Brown & Williams, 1975) and are incapable of judging their level of intoxication (Silverstein et al., 1974) indicates that there is a disorder in internal cue perception. Inasmuch as internal perception and attentional processes, as well as self-monitoring functions, are subserved by the anterior frontal region of the cerebrum, it can be concluded that the deficiency of alcoholics in performing these types of tasks is consistent with the hypothesis of a dysfunction in this brain region.

Several other neuropsychological impairments exhibited by alcoholics can also be interpreted as reflecting a prefrontal dysfunction. Deficits have been reported on tests requiring response persistence (Tarter, 1973), error utilization (Tarter, 1973), hypothesis testing (Klisz & Parsons, 1977), and visual search (Bertera & Parsons, 1978). The same types of impairments are also found in frontally lesioned patients (Crowne, 1983; Damasio, 1979). The observation that the magnitude of the deficits in alcoholics was either uncorrelated, or poorly correlated, with drinking history further suggests that the impairments, at least in part, preceded the alcoholism.

5.2 NEUROBEHAVIORAL CORRELATES OF THE LIMBIC AND DIENCEPHALIC REGIONS

These brain regions are primarily responsible for mediating emotional and motivational processes (Valenstein & Heilman, 1979). Anterior to the hypothalamus there is a neural circuit possessing primitive executive functions that are conditionable to both homeostatic states of the organism and environmental incentives (Panksepp, 1982). The primary function of this translimbic system is to initiate motor activity in preparation for consummatory behavior. In effect, this neural substrate comprises the anatomical basis for the psychological process of expectancy.

The "expectancy" neural system has been shown to play a major role in addiction (Panksepp, 1981). Indeed, expectancy variables are at least as influential as demographic variables in predicting adolescent drinking (Christiansen & Goldman, 1983), and appear to also play a major role in alcoholism (Higgins & Marlatt, 1973). At low doses, expectancy is probably more important than alcohol's pharmacological properties in determining the individual's emotional and motivational reactions (Wilson & Abrams, 1977).

Limbic and diencephalic mechanisms also regulate organismic arousal. Evidence for a dysfunction of arousal regulation mechanisms in persons at risk for alcoholism is derived from at least four sources. First, Mawson and Mawson (1977), in a comprehensive theoretical and empirical review, concluded that a neurotransmitter disequilibrium constitutes the neurobiological basis for psychopathy. Considering the similarities and associations between psychopathy and the type of alcoholism currently under discussion, it is reasonable to suspect that a similar neurochemical disorder underlies alcoholism. In addition, Schuckit et al. (1982) report low platelet MAO levels in high-risk individuals and low platelet MAO levels have been found in recovering alcoholics (Sullivan et al., 1978). Furthermore, low MAO levels have been found in suicidal alcoholics at autopsy (Gottfries, 1980). Finally, alcoholics

exhibit an information processing style that is characterized by stimulus augmenting (Fowler et al., 1980; Petrie, 1967; Schooler et al., 1978), which in turn is associated with low MAO levels (Buchsbaum & Ludwig, 1980; Von Knorring, 1976). The finding that the stimulus augmenting disposition is more characteristic of younger than older alcoholics (Brown & Cutter, 1977) and does not change with sobriety (Coger et al., 1976) also suggests, although does not prove, that this trait existed prior to the onset of alcoholism. Thus, there is some evidence to implicate a disruption of the monoaminergic system in persons predisposed to alcoholism, although in all likelihood the vulnerability involves more than just one neurotransmitter system.

Second, evidence for a disorder in arousal regulation has also been derived from psychophysiological research. Kissin and Hankoff (1959) reported that alcohol exerted a "normalizing" effect in alcoholics, suggesting that drinking may initially be prompted by arousal instability. The observation that alcoholics sustained a heart rate increment after the consumption of alcohol longer than nonalcoholics also points to a homeostatic disorder (Rosenberg & Buttsworth, 1969). In addition, Rubin et al. (1980) found evidence for arousal disturbances using pupillometric measures. Although possibly confounded somewhat by the effects of chronic drinking, the evidence nonetheless suggests the possibility of disrupted arousal mechanisms.

Third, alcoholics frequently abuse stimulants (including nicotine and caffeine), as well as tranquilizers and hypnotics (Kaufman, 1982). The motivation to use drugs may thus not be merely to achieve a desired pharmacological effect of either a high or low level of arousal, but rather may be aimed at attenuating arousal lability.

And fourth, behavioral investigations also implicate an impairment in arousal homeostasis. Prealcoholics have been found to be restless, unpredictable, and disinhibited — behaviors that suggest unstable and rapidly oscillating arousal states. Impulsivity, which is associated with anxiety (Gray et al., 1983), has also been reported to antedate alcoholism. Sensation seeking, reflecting a need to augment as well as optimize arousal states, is also correlated with alcohol use (Schwarz et al., 1978; Zuckerman et al., 1972). Emotional immaturity and moodiness, observed in preadolescent sons of alcoholics (Aronson & Gilbert, 1983), additionally suggest that there is dispositional lability in high-risk individuals, although the possibility remains that these disturbances may be due, in part, to the stress of living in a disrupted home environment. Furthermore, the demonstration that emotional excitability and neuroticism are predictive of subsequent drug abuse (Sieber & Bentler, 1982) and that alcoholics under 30 years of age are characterized by abnormally high levels of anxiety (Rosenberg, 1969) is congruent with the notion of a disruption of arousal-regulating mechanisms. And finally, neuroticism has been found to be characteristic of both alcoholics (Barnes, 1983) and sons of alcoholics (Barnes, 1983; Tarter et al., 1984) — a personality trait that Eysenck (1983) has argued develops from innate lability of the limbic system. Furthermore, body sway, which is positively correlated with measures of static ataxia in neurotic men but not in normals (Eysenck, 1947), is also found in persons at elevated risk for alcoholism (Lipscomb et al., 1979). Considered collectively,

therefore, the available evidence indicates that arousal instability characterizes prealcoholics, children at risk for alcoholism, and already affected alcoholics.

A limbic system involvement is also suggested by the particularly strong reinforcing effects that alcohol has in certain individuals. The medial forebrain bundle is integrally involved in mediating positive reinforcement. A differential reinforcing response to alcohol in high-risk individuals reported by Sher and Levenson (1982) therefore can be interpreted to suggest that alcohol exerts a particular effect on the neuronal substrate subserving reinforcement.

Preliminary evidence has also been obtained suggesting that a diencephalic disturbance, in conjunction with the endocrine system, may also be associated with the vulnerability to alcoholism. Proximal to the hypothalamus is the pituitary gland; thus a dysfunction in the diencephalon may also result in an endocrine-mediated disorder.

Tentative evidence suggests that this is the case. It has been found, for example, that delinquents who subsequently become alcoholic are more effeminate or "gynic" in their somatotype than individuals who do not develop alcoholism (Monnelly et al., 1983). Additional evidence that the vulnerability to alcoholism is related to certain morphological characteristics is provided in a study of 1,100 males conducted by Rees and Eysenck (1945). They found that 5.1% of "macrosomatics" used alcohol excessively, whereas only 1.9% of "microsomatics" and 3.0% of "mesosomatics" abused alcohol. This study demonstrates that as body size increases, so does the propensity for alcohol abuse. The same relationship also holds for moderate consumption levels. The micro-, meso-, and macrosomatics had a 44.2%, 45.2%, and 57.7% incidence of moderate alcohol consumption pattern.

A study performed by Seltzer (1946) on 258 Harvard University students also merits consideration. Subjects who had body disproportions (e.g., thin legs and wide shoulders) were characterized by autonomic instability, mood fluctuations, poor capacity to make an easy social adjustment, inadequate personality integration, and hypersensitivity. This study is noteworthy in that these psychophysiological characteristics have been linked to the vulnerability to alcoholism (Tarter et al., 1985). Whether these types of body disproportions are found in persons at high risk remains, however, to be determined.

In another investigation, Halberg (personal communication) found that the circannual levels of plasma DHEA-S in high-risk individuals were both lower and more variable than in those at low risk for alcoholism and drug abuse. This preliminary finding suggests that endocrine factors may precede the onset of alcoholism, but their role in neurochemical and somatic processes still needs to be elucidated.

5.3 NEUROBEHAVIORAL CORRELATES OF THE MIDBRAIN REGION

Among the functions primarily subserved by the midbrain region are consciousness, attention, and arousal. To date, there have been no neurobehavioral studies specifically directed to elucidating midbrain functioning in prealcoholics or in persons at risk for developing alcoholism. However, deficient attention and concentration processes appear to characterize such

persons (Wender et al., 1981; Wood et al., 1976, 1983) and thus by inference implicate the midbrain reticular activating system. As previously discussed, hyperactivity is one of the components of the vulnerability. This disorder is frequently associated with an attention deficit disorder (American Psychiatric Association, 1980), as well as a disturbance in arousal regulatory mechanisms (Denckla & Heilman, 1979; Douglas, 1980). That approximately one out of three alcoholics meets the criteria for an attention deficit disorder, residual type, suggests that the attention problems persist into adulthood and augment the risk for becoming alcoholic (Wood et al., 1983). Pupillometric studies of alcoholics also point to a disturbance in midbrain mechanisms (Rubin et al., 1977, 1978, 1980).

Alcoholics enhance or magnify sensory input. Either employing the kinesthetic aftereffect test (Petrie, 1967) or measuring brain evoked potentials (Buchsbaum & Ludwig, 1980; Coger et al., 1976) reveals that alcoholics are "stimulus augmenters." Inasmuch as the reticular formation is integrally involved in modulating and controlling stimulus input (Hernandez-Peon, 1961), it can be concluded that the risk for alcoholism is, to some extent, related to the neurophysiological activity of this neural network.

6. PRACTICAL IMPLICATIONS

If subsequent research confirms the theory that neuropsychological characteristics are associated with the risk for alcoholism, it not only would substantially increase our capacity to identify potential alcoholics but also would enable the application of specific prophylactic interventions. For instance, the capacities subserved by the anterior frontal region could be targeted for developmental monitoring. If intervention is at some point deemed desirable, the child could then be taught rule-learning strategies to enhance cognitive skills or be placed in a behavioral modification program to reduce behavioral problems stemming from a dysfunction of this brain region. The fact that the anterior brain regions do not functionally mature until adolescence (Luria, 1961, 1966; Vygotsky, 1962) suggests that deficits in high-risk children may not be observed until then. In other words, preadolescent high-risk children may not be distinguishable from other children. The prodromal signs of a negative outcome are, nonetheless, still expressed at a young enough age to be treated prior to the onset of problematic drinking in most cases. Thus, primary prevention strategies, having circumscribed and well-defined cognitive and behavioral change objectives, could be applied to high-risk individuals.

Other practical ramifications of the theory pertain to the affective components of the vulnerability. For example, the hypothesized disruption of homeostatic mechanisms, resulting in emotional lability and anxiety, may be amenable to such interventions as biofeedback, which emphasizes self-regulation of physiological activity.

The hypothesis that a neurochemical disturbance is responsible for the

difficulties in arousal regulation would also suggest the possible value of pharmacological interventions. The demonstration that certain drugs such as lithium (Merry et al., 1976), methylphenidate (Wood et al., 1976), and pemoline (Wood et al., 1976) are therapeutically beneficial for some alcoholics is in agreement with the theory advanced herein that arousal dysregulation and a disturbance of the monoaminergic system are associated with the vulnerability to alcoholism.

7. RELATIONSHIP TO OTHER THEORIES

To briefly recapitulate: The theory advanced herein argues for an inherited neurological disturbance of neural systems coursing along the frontal-midbrain axis. This disturbance is manifest in a range of cognitive, affective, and behavioral processes that are known to be subserved by this brain region. Intervening between the anatomical substrate and behavior is a genetically determined neurotransmitter disturbance of mechanisms responsible for the regulation of organismic arousal. Though this neurobehavioral theory is somewhat speculative, owing primarily to the dearth of experimental evidence, it nonetheless is able to accommodate much of the existing evidence regarding the vulnerability to alcoholism.

From material reviewed elsewhere, little doubt can be harbored regarding the genetic predisposition to alcohol (Goodwin, 1983; Grove & Cadoret, 1983). The phenotypic expression of the genetic diathesis has not, however, been systematically studied in humans. Previous reviews by the authors have argued for the heritability of certain behavioral characteristics that, if present, increase the risk for alcoholism (Alterman & Tarter, 1983). The present discussion extends this line of reasoning by demonstrating that the psychological evidence can be organized into a neurobehavioral framework. The inheritance of cognitive capacities is well documented, and hence the thesis contained herein, by addressing the question of *what* is inherited, can be considered to comprise a neurobehavioral-genetic theory.

Insofar as behavioral processes are the final link in the chain of biological mechanisms, there are other intervening levels of biological analysis that potentially may also reflect the vulnerability. In various places in this discussion, we have raised the possibility that certain neurochemical and physiological disturbances are associated with the risk for alcoholism, as well as for the manifest neuropsychological deficits. A comprehensive discussion of neurochemical mechanisms can be found in Chapter 2, by Tabakoff and Hoffman. It will be seen that an extensive literature has accrued; however, most of what is currently known is derived from investigations with animals. This discussion, by focusing on psychological processes, underscores the need to conduct investigations in which behavioral and biochemical variables are conjointly manipulated and measured.

The proposed neuropsychological theory is also compatible with social learning and conditioning perspectives of alcoholism etiology. While this

chapter addressed the interrelationship between the anatomical substrate and cognitive, emotional, and motivational processes associated with alcoholism vulnerability, the importance of learned patterns of behavior and cognition was also considered in the context of the neural system mediating expectancy. These learned patterns of behavior are, however, theorized to be predisposed in vulnerable individuals. Craving for alcohol and loss of control over drinking are hypothesized to involve the interaction between cognitive and physiological processes (Ludwig, 1983). Clarifying the role and functions of the "expectancy" neural system may therefore contribute to a better understanding of the factors underlying these symptoms, especially since expectancy in psychological research has been shown to influence their occurrence in alcoholics. Moreover, the range of psychological processes subserved by the anterior-basal system raises other important questions pertinent to social learning and conditioning theories of alcoholism. For example, how does the implicated disturbance in arousal affect conditionability? And how does the hypothesized inability to self-monitor behavior relate to the capacity to acquire social skills? Thus, the present theory affords the opportunity to relate a variety of manifest behavioral characteristics associated with the propensity for alcohol abuse and alcoholism to a dysfunctional brain system.

It is interesting to note that personality characteristics and emotional disturbances can also be investigated from a neurobehavioral perspective (Eysenck, 1967; Zuckerman, 1983). Certain personality factors, as discussed by Cox in Chapter 6, as well as by Barnes (1983), suggest that these psychological parameters may also contribute to the risk for alcoholism. A potentially fruitful area of research would therefore involve the integration of neuropsychological and personality measurement techniques in elucidating alcoholism vulnerability.

Other theoretical perspectives of alcoholism etiology are not capable of being readily integrated into a neurobehavioral analysis. Part of this problem stems from the nature of the constructs thought to be intrinsic to alcoholism etiology. For example, the language of discourse of humanistic psychologists and psychodynamic theorists cannot easily be operationalized and linked to biological processes. Also, macrosystem factors, such as political and economic controls over beverage distribution, do not directly bear on the theory described in this chapter. These latter approaches are more concerned with regulating drinking practices of an entire population, rather than being concerned with alcoholism vulnerability in a particular segment of society.

8. CONCLUDING COMMENTS

It was postulated that a dysfunction of the anterior-basal system of the brain underlies the vulnerability to alcoholism. Whereas neuropsychological studies of alcoholics have suggested that a disturbance to this brain system may be consequent to a history of chronic drinking (Tarter, 1975), it is theorized herein that an inherited dysfunction to this brain region may also precede

alcoholism in certain vulnerable individuals.

The present discussion constitutes the first systematic attempt to advance a neuropsychological theory of alcoholism etiology. Not surprisingly, therefore, the information is limited, and much of what is known was originally acquired in order to address issues that were not neuropsychological. Nonetheless, the bulk of available evidence obtained from individuals at high risk for alcoholism, from prealcoholics, and from already affected alcoholics is consistent with the type of impairments that are expected to be found in individuals with an anterior-basal dysfunction.

Despite the dearth of direct empirical investigations, the convergence of diverse findings from different types of populations tentatively confirms the neuropsychological theory implicating a dysfunction of neural systems coursing along the frontal-midbrain axis. At this stage of research, however, the theory should be viewed as a heuristic perspective from which further neuropsychological investigations can proceed in a logical and systematic fashion.

Thus, in summary, the theory awaits further testing prior to its confirmation or disconfirmation. The present review and integration of the literature have demonstrated the value of a neuropsychological approach. Hopefully they will spur other researchers to participate in the difficult task of delineating the neurobehavioral characteristics associated with alcoholism vulnerability, as well as the impact of these characteristics on outcome in adulthood.

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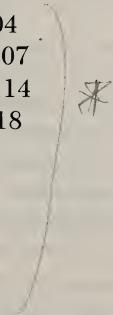
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4. PSYCHOANALYTIC THEORY OF ALCOHOLISM

Herbert Barry, III

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1. INTRODUCTION

Psychopathology is emphasized in the comprehensive explanation of human behavior that is attempted by psychoanalytic theory. This emphasis is in accordance with the origin of the theory as a therapeutic technique. The normally adaptive motive to seek pleasure and avoid pain is distorted in pathological behavior, which originates in environmental conditions that blocked normal personality development during infancy and early childhood.

Alcoholism is defined as repeated intake of alcoholic beverages in quantities large enough to cause severe physical or social damage to the drinker. The voluntary consumption of alcohol is originally motivated by a desire for its pleasurable effects. For some people the drinking becomes a repetitious, self-destructive behavior. Because of this transition from pleasure to pathology, alcoholism is a behavior with special pertinence for psychoanalytic theory and therapy. The theory suggests that we search for disturbances of personality as sources of the subsequent pathological behavior of excessive drinking.

Two contrasting explanations of alcoholism are consistent with psychoanalytic theory. According to one explanation, the individual finds the effects of alcohol intoxication to be unusually pleasurable. According to the other explanation, the individual is deficient in avoiding the painful consequences of

alcoholism. Both explanations might contribute to the alcoholism of the same individual.

A person who experiences unusually strong pleasurable effects from alcohol might drink excessively even when the effects become destructive. The pleasurable effect is usually attributed to relief from anxieties and conflicts rather than a direct sensuous satisfaction. This mechanism for pleasure is consistent with the predominantly depressant effect of intoxicating doses of alcohol.

A high dose of alcohol has aversive effects, which include loss of capabilities while intoxicated and a hangover afterward. More severe damage develops from repeated intake of high doses. A drinker with deficient avoidance of these effects would lack the protection most people have against alcoholism.

According to psychoanalytic theory, adaptive behavior requires harmonious functioning of three components of the self (ego, id, superego). The functions of these components change during four successive stages of psychosexual development (oral, anal, phallic, genital). Alcoholism and other pathological behaviors are attributed to disturbances in these stages of development, resulting in destructive interactions among the three components of self.

The psychoanalytic theory described in this chapter is based on the writings of Sigmund Freud and his orthodox followers. This chapter briefly differentiates psychoanalytic theory from rival theories of personality that emphasize social conditions (Alfred Adler) or cultural symbols (Carl Gustav Jung).

Among other theories of alcoholism, biological theories are especially pertinent because of their alternative explanations for vulnerability of some individuals but not others. These different theories are not mutually exclusive. Psychoanalytic theory can be supplemented by analysis of biological differences in vulnerability; the biological theories can be broadened by analysis of developmental and motivational attributes that induce alcoholism.

2. PRINCIPAL ELEMENTS OF THE THEORY

Psychoanalytic theory identifies three main sources of maladaptive behavior: (1) seeking sensuous satisfaction; (2) conflict among components of the self; (3) fixation in the infantile past. Each of these elements contributes to the development of alcoholism.

2.1 SEEKING SENSOUS SATISFACTION

The primary motive for behavior is to enjoy physical gratification. The "libidinal" satisfaction referred to by Freud constitutes a constellation of sensual drives rather than merely the genital sexual behavior that is often regarded as the primary pleasure according to psychoanalytic theory. Deprivation of this sensuous satisfaction impels the craving for alcohol or for other substitute sources of pleasure.

One form of sensuous satisfaction is relief from anxiety. The need to escape from physical or emotional pain can explain alcohol intoxication and

other behaviors that are physically harmful rather than beneficial. Psychoanalysts and other therapists treat many patients who seek to escape from an anxious, miserable life. Behaviors that cause distress, such as obsessive-compulsive rituals and phobias, may be adaptive if they relieve more severe anxiety.

Alcohol is a toxic agent that provides relief and thus pleasure. Intoxication impairs the functioning of the central nervous system and thereby diminishes capabilities. This detrimental effect is sought because alcohol intoxication also diminishes feelings of tension and anxiety. A disinhibitory effect of alcohol contributes to the pleasurable effect of intoxication.

2.2 CONFLICT AMONG COMPONENTS OF SELF

The self is divided into three components: id, superego, and ego.

The id is the instinctual striving for pleasure or relief. It demands gratification urgently and immediately regardless of the realities of the situation. Alcohol intoxication can provide this gratification.

The superego is the conscience, the ethical component derived from relationships with parents and other social agents. The aims of the superego and id are usually opposed to each other. This conflict is often intense because both opposing forces resemble each other in their demand for immediate gratification without regard for each other or for external reality. The conflict induces anxiety; inability to satisfy both opposing demands is frustrating. Alcohol can relieve both the anxiety and the frustration.

The ego represents the organizing, coping function. It tries to reconcile the id and superego with each other and with external reality. This rational, self-conscious component of the self is expressed in the alcoholic's attempts to control the excessive drinking.

Table 1 identifies the present author's view of the conditions of each component in sober and intoxicated nonalcoholics. The id of the sober alcoholic craves for relief that is obtained by alcohol intoxication.

Defence mechanisms are induced by the conflict among components of the self to protect the coping function of the ego. Repression from awareness is the most direct defence mechanism, such as denial of the id desires or, alternatively, denial of the opposing superego desires. A frequent form of repression is denial of reality, for example the alcoholic's denial of the self-destructive effects of drinking. More complex defence mechanisms include reaction formation, such as avoidance of drinking, and displacement, such as addiction to other drugs or to gambling. Another defence mechanism is identification with the parents, incorporating superego demands into the ego. This is a source of conflict because of antagonistic feelings toward these authority figures. Disturbances of this identification disrupt the social and sexual relationships of many alcoholics.

Psychopathology. Failure of the ego to reconcile the conflict between id and superego is the source of the two major types of pathology, neurosis and psychosis. In neurosis, the ego is intact but disrupted so that behavior is maladaptive and inconsistent. Occasional severe alcohol intoxication may be a neurotic symptom. Another neurotic symptom is rigid renunciation of pleasures, including alcoholic beverages. In psychosis, the ego is overwhelmed and

TABLE 1

**Conditions of the three components of self
are summarized in nonalcoholics and in alcoholics
when they are sober and intoxicated**

	NONALCOHOLICS		ALCOHOLICS	
	<i>Sober</i>	<i>Intoxicated</i>	<i>Sober</i>	<i>Intoxicated</i>
<i>Id</i>	Striving	Disinhibited	Craving	Triumphant
<i>Superego</i>	Restraining	Weakened	Punishing	Disrupted
<i>Ego</i>	Controlling	Exhilarated	Anxious	Overwhelmed

its function is destroyed; examples are schizophrenia and chronic depression. Excessive alcohol intake is a self-destructive behavior that sometimes accompanies psychotic illness.

2.3 FIXATION IN THE INFANTILE PAST

The human being is born in a primitive, undeveloped state. Freud identified four successive stages of development: oral, anal, phallic, and genital.

Each of the first three stages designates an immature response to conflicts among the three components of the self (id, superego, ego). Excessive gratification or frustration at any of these stages interferes with the transition to the next stage, and results in fixation at an immature stage. Regression to an earlier stage occurs under the combined influences of frustration at a later stage and partial fixation at the earlier stage.

Fixation and regression indicate disturbances of adaptive behavior. The ego functioning is primitive or disrupted. This situation induces maladaptive behavior, including alcoholism.

Oral stage. The id is dominant at this infantile stage, prior to development of the superego and ego. The id demands the instinctual pleasures, such as drinking, eating, sleep, and warmth. Alcohol is an attractive substance for people who are fixated at the oral stage: the beverage is taken by mouth, is a liquid and food, is a sedative, and induces a feeling of warmth.

Anal stage. Training in bowel and bladder control constitutes an early confrontation of id with superego forces, thereby developing self-restraint and inducing rebellion against control. Ambivalent feelings and ritualistic repetitions of behavior originate in this stage. Many alcoholics display anal attributes in their ambivalence toward alcohol intoxication: repeated, compulsive drink-

ing alternates with guilt feelings and compulsive efforts to abstain.

Phallic stage. Feelings toward the parents threaten development of the capability for heterosexual love. In the Oedipus conflict, which was emphasized by Freud, the boy's libidinal desire for his mother establishes his father as a hated and dangerous rival. (Freud emphasized this pattern, named after a king in ancient Greek mythology who unknowingly killed his father and married his mother.) For a girl, the corresponding Electra conflict opposes love for her father with hatred and fear toward her mother. Because of these conflicts, heterosexual attachments are accompanied by fear, jealousy, and violence. Alcohol intoxication weakens the adaptive restraints against this conflict. Alcoholism therefore induces the maladaptive behavior of avoiding or attacking sexual partners or seeking sexual partners who are taboo or otherwise inappropriate.

Genital stage. This is the stage of maturity, in which the ego controls the id and the superego. The libidinal drives and the conscience are thereby made compatible with each other and with reality. This adaptive stage of development is indicated by mutual mature love with a heterosexual partner. The fortunate people who attain this final stage enjoy healthy adjustment and are effectively protected against alcoholism.

Multiple fixations. Human beings are complex and capable of logically inconsistent behaviors. When the preponderant behavior progresses to a more mature stage, some components of behavior remain in the earlier stage. Regression to an earlier stage does not mean complete loss of the more developed behavior; the different stages coexist and compete. Symptoms of alcoholism constitute a mixture of oral passivity, anal compulsiveness, phallic aggressiveness, and efforts to attain genital maturity.

3. FREUD'S COMMENTS ON ALCOHOLISM

Psychoanalytic theory originated with one man, Sigmund Freud. Although he made some comments about alcoholism, Freud was more concerned with other types of psychopathology. Therefore, the principal applications of the theory to alcoholism were by his followers. Extensive information on Freud and on his theory is available in a three-volume biography (Jones, 1953-57). A later book by Roazen (1971), based on interviews with many adherents and other acquaintances of Freud, contains much further information and excellent critical comments on Freud and on psychoanalytic theory.

The few published comments by Freud on alcohol and on alcoholism have historical interest. Most of these passages express his personal attitudes, rather than formal theoretical statements, but all of Freud's writings influenced his followers and thus constitute important background to the more extensive writings on alcoholism by other psychoanalysts.

Most quotations of Freud's writings below are from *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, translated and edited by James Strachey, in 24 volumes (Strachey, 1966-74). Volume 24 includes a

subject index that aided in the search for comments on alcohol. The publication dates of the articles or books indicate when they became available to his followers. Some quotations are from the complete letters of Freud to Fliess (Masson, 1985). These were published recently and thus could not have influenced prior writers. They provide further information, however, about Freud's attitudes and drinking behavior in an informal and self-revealing context.

3.1 EARLY WRITINGS BY FREUD

Alcoholism was mentioned with relatively high frequency in Freud's early writings, before his theory was well developed or widely accepted. Many of these early passages describe alcohol abuse as only one of several toxic influences. Excessive drinking is mentioned as an example of conditions that can aggravate neurotic or psychotic tendencies.

The first example is from an article "Hysteria" published in 1888, when Freud was 32 years old (Strachey, vol.1, p.50).

As factors which produce outbreaks of acute hysterical illness may be adduced: trauma, intoxication (lead, alcohol), grief, emotion, exhausting illness — anything, in short, which is able to exercise a powerful effect of a detrimental kind.

Another passage occurs shortly after (p.51):

Conditions brought about by severe general trauma...are regarded as hysteria by Charcot.... Charcot has also proved that encephalopathy from plumbism is related to hysteria and also that anaesthesia which is common in alcoholics is not a separate illness but a symptom of hysteria.

The theme of alcoholism summing with other pathological factors is expressed in an article "On the Grounds for Detaching a Particular Syndrome from Neurasthenia under the Description 'Anxiety Neurosis'." It was published in 1895 (Strachey, vol.1, p.106).

...a chronic alcoholic will in the end develop a cirrhosis or some other illness, or will, under the influence of a fever, fall a victim to delirium.

This theme is elaborated in "A Reply to Criticisms of my Paper on Anxiety Neurosis" published later in 1895 (Strachey, vol.1, p.130):

It will readily be granted that there are aetiological factors which, in order to exercise their effect, must operate with a certain intensity (or quantity) and over a certain period of time — which, that is to say, become summated. The effects of alcohol are a standard example of causation like this through summation.

The next four quotations are from Freud's letters to Wilhelm Fliess. These extracts were first published in English in 1954. The complete letters were published subsequently (Masson, 1985). The intense friendship with Fliess, a contemporary physician, was an important influence on Freud's development of psychoanalytic theory.

In Draft H (Strachey, vol.1, p.210), sent on 24 January 1895, Freud introduced a link between alcoholism and sexual jealousy:

The alcoholic will never admit to himself that he has become impotent through drink. However much alcohol he can tolerate, he cannot tolerate this piece of knowledge. So his wife is to blame — delusions of jealousy and so on.

Subsequent writings on this idea, published much earlier, were in 1900

(Strachey, vol.4, p.89) and in 1911 (Strachey, vol.12, p.64).

Draft K (1 January 1896) describes symptoms of obsessional neurosis (Strachey, vol.1, p.225). Alcoholism is portrayed as a substitute for frustrated primary gratifications:

Other secondary symptoms arise if the compulsion is transferred to motor impulses against the obsession — for instance, to brooding, drinking (*dipsomania*), protective ceremonials, *folie du doute*.

A case of dipsomania, beginning in a man at the age of 50 years, was attributed to substitution for a homosexual perversion (Letter 55 to Fliess, 11 January 1897). The uncle who seduced a male patient of Freud was described as follows:

...a man of genius who, however, had attacks of the severest dipsomania from his fiftieth year onward. These attacks regularly started with catarrh and hoarseness (the oral sexual system!) — that is, with the reproduction of his own passive experiences. Now, until he fell ill himself, the man had been a pervert and consequently healthy. The dipsomania arose through the strengthening — or rather through the *substitution* of the one impulse for the associated sexual one.

Addictions to alcohol, morphine, tobacco, etc. are described as replacements for the “primal addiction” of masturbation (Letter 79 to Fliess, 22 December 1897: Strachey, vol.1, p.272).

It has dawned on me that masturbation is the one major habit, the “primal addiction” and that it is only as a substitute and replacement for it that the other addictions — for alcohol, morphine, tobacco, etc. — come into existence.

3.2 LETTERS TO FLIESS

The complete letters to Fliess, in Masson (1985), have revealed further writings Freud did not intend to be published. The following quotations are his comments on alcohol or alcoholism. These references to alcohol, and especially to his own use of it, may give us further insight into Freud's views and the circumstances of their development.

One of the early letters, on 12 July 1892 (Masson, p.32), stated that the opportunity to select from the library of the recently deceased Dr. Meynert was “somehow like a savage drinking mead from his enemy's skull.”

In a letter on 21 May 1894 (p.75), on a patient with neurasthenic dyspepsia, “He himself ascribes the cause of his trouble...to his having worked, drunk, and smoked heavily...”

When Freud felt sick after watching heavy bleeding from the nose of a patient, a woman “then brought me a small glass of cognac and I became myself again” (letter of 8 March 1895: p.117).

On 16 March 1896 (p.178) Freud stated that in a forthcoming meeting he would bring to Fliess “a case of dipsomania, which resolved itself very obviously according to my schema.”

Freud commented that “any drop of alcohol makes me completely stupid” on 16 April 1896 (p.181). A letter on 26 April 1896 (pp.183-184) contained an anecdote in which a doctor tells an old bon vivant: ‘Dear friend, from now on, no more wine, women, and song,’ to which the latter replies, ‘Very well, I'll stop *singing*.’

On 17 December 1896 (p.218) Freud mentioned a case in whom “the reluctance to drink beer and to shave was elucidated by a scene in which a nurse sits down...[with bare buttocks] in a shallow shaving bowl filled with beer in order to let herself be licked...”

A personal drinking situation was mentioned on 10 March 1898 (p.302): “My seminar was particularly lively this year...when the university was closed, I went on lecturing in my room over a mug of beer and with cigars.”

A comparison with an alcoholic beverage was suggested in references to “a draft” of a “punch made of Lethe” (the river of forgetfulness) in a letter on 6 September 1897 (p.263) during a trip to Italy and again in a letter on 12 December 1897 (p.286) looking forward to a meeting with Fliess.

A letter on 14 April 1898 contained several references to drinking (pp.308-309). Freud described an ancient Roman statue of “Priapus as an old man, whose genitals are being covered by a salinus and who henceforth can give himself over to drink....” After Freud had “a choice Istrian wine” with a meal, “A modern drunk was lying on the ancient paving stones” of an old Roman road. In a visit to a cave the guide was “in a deep alcoholic stupor, but completely surefooted, and full of humor.” Freud overtipped him “so that he can drink his life away faster.”

In a letter on 5 December 1898 (p.335): “Occasionally I have longed for a strong and sweet drop of the juice of grapes — even if it cannot be a ‘punch made of Lethe’ — but I was ashamed of acquiring a new vice.”

On 16 January 1899 (p.340) Freud was feeling miserable and “seeking restoration in a bottle of barolo.... All sorts of comfort come from the wine....”

On 19 February 1899 (p.345), likening his preoccupation with work to a neoplasm, “The neoplasm in its most recent stages of development likes to drink wine.”

The following comments were on 24 April 1899 (p.350):

Thanks for your announcement of the nectar.... Since drinking alone is merely a vice, you will permit me to empty a glass apiece for Wilhelm, Ida, Robert, and Paulinchen.... Therefore, I gladly will drink the punch made of Lethe. Alexander has returned, he too saturated with wine.

On 25 May 1899 (p.351): “I still carry in my pocket the instructions on how to handle the ‘wine of the gods’....” A few days later, on 28 May 1899 (p.353): “the wine has arrived and is resting, according to your directions....”

A letter on 16 June 1899 (p.355) referred to an expected child conceived by Fliess during a trip in Italy and a gift of Marsala wine.

...some of the fire of Italian wine will circulate in his veins.... The heavenly marsala is already on our table, but we drink it only in drops; Martha counted the bottles and took charge of them lest in my loneliness I succumb to the consolation of drink.

On 27 June 1899 (p.357): “I am gradually becoming accustomed to the wine; it seems like an old friend. I plan to drink a lot of it in July.”

On his work on *The Interpretation of Dreams* on 8 July 1899 (p.359): “I cannot manage more than two hours a day without calling on Friend Marsala for help. ‘He’ deludes me into thinking that things are not really so bleak as they appear to me when sober.”

On 11 March 1900 (p.404): "You know how limited my pleasures are... alcohol does nothing for me...."

A letter on 7 August 1901 (p.447), during vacation, contained the comment "I have a hunch that it will not be possible to do without eight to twelve days of olive oil and wine."

3.3 MATURE WRITINGS BY FREUD

The major books and articles that developed psychoanalytic theory contain only a few mentions of alcoholism. Some of the ideas expressed briefly by Freud were elaborated by his followers.

The highly influential and admired book *The Interpretation of Dreams* (1900) suggested the association of alcoholism with sexual jealousy, which is prominent in later writings by Freud's followers, in a passage preceded by examples of pathological symptoms or psychosis limited to dream-life (Strachey, vol.4, p.89):

Similar observations are reported by de Sanctis [1899, 226] (a dream of an alcoholic patient which was equivalent to a paranoia, and which represented voices accusing his wife of unfaithfulness)...

In "Three Essays on the Theory of Sexuality" (1905), drinking is one of the examples of addictive behavior in a passage on auto-erotic sucking by children "in whom there is a constitutional intensification of the erotogenic significance of the labial region" (Strachey, vol.7, p.182):

If that significance persists, these same children when they are grown up will become epicures in kissing, will be inclined to perverse kissing, or, if males, will have a powerful motive for drinking and smoking.

Alcoholism is one of the examples in an article "Psychical (or Mental) Treatment," published in 1905 (Strachey, vol.7, p.299). Freud pointed out the usefulness of hypnotism for breaking morbid habits "such as alcoholism, morphine addiction, or sexual aberrations."

One of Freud's longest passages on alcohol reproduces a joke that portrays drinking as an escape from unpleasant reality. This was one of the examples in his book *Jokes and Their Relation to the Unconscious*, published in 1905 (Strachey, vol.8, pp.113-114). The story and Freud's concluding comment express well his recognition of the dangers and also the attractions of drinking.

A doctor correctly diagnosed a man's deafness as due to drinking too much brandy. The patient recovered from his deafness when he followed the doctor's advice to stop drinking brandy. Subsequently, the patient resumed drinking and became deaf again. He explained to the doctor, "I was able to hear. But nothing I heard was as good as the brandy." Freud added "the sad question: may not the man have been right in his choice?"

Freud contrasted the sexual instinct with addiction to alcohol in "Contributions to the Psychology of Love." This article was published in 1910 (Strachey, vol.11, p.188). The preceding passage had pointed out the fickleness that is typical of erotic love.

Is it not true that wine always provides the drinker with the same toxic satisfaction, which in poetry has so often been compared to erotic satisfac-

tion — a comparison acceptable from the scientific point of view as well?...habit constantly tightens the bond between a man and the kind of wine he drinks.

The theme of alcoholism as a defence against repressed homosexual desires is expressed in "Psycho-analytic Notes on an Autobiographical Account of a Case of Paranoia (Dementia Paranoides)." This is the famous case of Dr. Schreber, published in 1911 (Strachey, vol.12, p.64). Freud described the role of alcohol in men's delusions of jealousy, although alcoholism was not one of Dr. Schreber's symptoms.

We know that that source of pleasure removes inhibitions and undoes sublimations. It is not infrequently disappointment over a woman that drives a man to drink — but this means, as a rule, that he resorts to the public-house and to the company of men, who afford him the emotional satisfaction which he has failed to get from his wife at home.... he suspects the woman in relation to all the men whom he himself is tempted to love.

The next reference to alcoholism is in "A Metapsychological Supplement to the Theory of Dreams" in 1917 (Strachey, vol.14, pp.233-234). The "wishful psychosis of amentia" is described as "the reaction to a loss which reality affirms, but which the ego has to deny, since it finds it insupportable." A footnote to this passage displays Freud's impressive understanding of medical symptoms. He recognized that delirium tremens constitutes an alcohol withdrawal reaction many years before this was scientifically demonstrated and generally acknowledged:

I may venture to suggest in this connection that the toxic hallucinations, too, e.g. alcoholic delirium, are to be understood in an analogous fashion. Here the unbearable loss imposed by reality would be precisely the loss of alcohol. When the latter is supplied, the hallucinations cease.

Another publication in the same year (1917) stated the idea that the pleasurable effect of intoxication from alcohol is attributable to denial of opposite, unpleasant feelings. This is in "Mourning and Melancholia" (Strachey, vol.14, p.254).

Alcoholic intoxication, which belongs to the same class of states, may (in so far as it is an elated one) be explained in the same way; here there is probably a suspension, produced by toxins, of expenditures of energy in repression.

The elated feeling induced by intoxication is thus attributed to a dangerous rejection of reality. Later in the same article (Strachey, vol.14, p.258), Freud suggested that the transition from melancholia to mania "must be linked with regression of the libido to narcissism." This expresses a further unfavorable view of the elation caused by intoxication.

3.4 LATE WRITINGS BY FREUD

An unusually lengthy comment on drug intoxication was included in *Civilization and Its Discontents*, published in 1930, when Freud was 74 years old (Strachey, vol.21, p.78). This passage elaborated his earlier theme of alcohol as a means of warding off unpleasant feelings. Freud's understanding and disapproval of the attraction of intoxicating substances included remarkable anticipation of findings, many years later, that endogenous substances (designated as "endocoids" by Lal et al., 1985) can reproduce the effects of an

exogenous drug.

I do not think that anyone completely understands its mechanism, but it is a fact that there are foreign substances which, when present in the blood or tissues, directly cause us pleasurable sensations; and they also so alter the conditions governing our sensibility that we become incapable of receiving unpleasurable impulses. The two effects not only occur simultaneously, but seem to be intimately bound up with each other. But there must be substances in the chemistry of our own bodies which have similar effects, for we know at least one pathological state, mania, in which a condition similar to intoxication arises without the administration of any intoxicating drug. Besides this, our normal mental life exhibits oscillations between a comparatively easy liberation of pleasure and a comparatively difficult one, parallel with which there goes a diminished or an increased receptivity to unpleasure. It is greatly to be regretted that this toxic side of mental processes has so far escaped scientific examination. The service rendered by intoxicating media in the struggle for happiness and in keeping misery at a distance is so highly prized as a benefit that individuals and peoples alike have given them an established place in the economics of their libido. We owe to such media not merely the immediate yield of pleasure, but also a greatly desired degree of independence from the external world. For one knows that, with the help of this "drowner of cares" one can at any time withdraw from the pressure of reality and find refuge in a world of one's own with better conditions of sensibility. As is well known, it is precisely this property of intoxicants which also determines their danger and their injuriousness. They are responsible, in certain circumstances, for the useless waste of a large quota of energy which might have been employed for the improvement of the human lot.

Another passage on alcoholism, later in the same book (Strachey, vol.21, p.84), is a succinct summary of Freud's recognition of the attractions and dangers of alcohol intoxication.

The man who sees his pursuit of happiness come to nothing in later years can still find consolation in the yield of pleasure of chronic intoxication; or he can embark on the desperate attempt at rebellion seen in a psychosis.

The year before his death, Freud reiterated the idea of alcoholism as a dangerous source of relief in "Comment on Anti-Semitism." This was a brief article (Freud, 1938) stating a "Gentile's" view of ways in which Jews are superior. One of the items of Jewish superiority is that "They do not need so much alcohol as we do in order to make life tolerable...." This is not included in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*. It is quoted by Roazen (1971, p.540) and cited by Jones (1957, vol.3, p.540).

3.5 CONCLUSIONS ABOUT FREUD'S COMMENTS

The pathological behavior of excessive drinking is explained by unusual need for the pleasurable effect. The pleasure is attributed to relief from misery rather than to enhancement of normal sensuous pleasures.

Fixation in the oral stage is emphasized in Freud's comments on gratification of oral desires as the motive for drinking. A conflict between id and superego in the phallic stage is indicated by alcohol intoxication in men as a response to homosexual desires, repressed from conscious awareness. These comments by Freud are notable because they were developed by some of his

followers.

Alcohol is seldom mentioned in Freud's voluminous publications. This seems to indicate an inhibited rather than weak interest in alcohol. The few comments on alcohol described vividly its attractions and dangers, in addition to impressive displays of scientific and medical understanding.

The more numerous references to drinking and alcoholism in the complete letters to Fliess (Masson, 1985) provide further evidence for avoidance of the topic rather than indifference to it in most of Freud's publications. Descriptions of yearning for wine and frequent drinking in some of the letters in 1899 coincided with completion of Freud's first major contribution to psychoanalytic literature, *The Interpretation of Dreams*. The decline of their friendship beginning in 1900 (Masson, 1985) was at least partly attributable to effects of this major achievement on both men.

Freud drank lightly according to most of the biographical information. (This is consistent with the prevalent Jewish practice that has been documented by Snyder, 1958.) Some items, however, indicate intense but rigorously controlled attraction to alcohol intoxication. Freud persuaded Jung to abandon teetotalism (Roazen, 1971, p.246) although Freud disliked "the faint mental obfuscation that even a slight drink induces" (Jones, 1953-57, vol.3, p.386). Freud explained in a letter to a friend that a contributory cause of several fainting attacks was "often a bit of alcohol for which I have no tolerance" (Roazen, 1971, p.249).

Use of other drugs was prominent in Freud's personal history. In the complete letters to Fliess (Masson, 1985) Freud reported using cocaine and repeated efforts to abstain from smoking. He continued to smoke heavily throughout the remainder of his life, even while dying of oral cancer. Some of his comments on alcohol are also pertinent to these other addictive substances.

4. DEVELOPMENT OF THEORY BY OTHERS

In contrast to Freud's few statements about alcoholism, some of his close associates made extensive applications of psychoanalytic theory to alcoholism. Other writers have extended his theory in ways he might not have approved totally.

Crowley (1939) summarized writings by several psychoanalytic theorists. Lorand (1945) contributed a brief review. Alcoholism is prominent in the systematic, comprehensive exposition of psychoanalytic theory by Fenichel (1945). Reviews by Blum (1966) and Blum and Blum (1967) applied psychoanalytic theory to therapy of alcoholics.

Two main types of explanations for alcoholism were developed by Freud's followers. One type of explanation is based on an unusually strong pleasurable effect of alcohol intoxication, the pleasure being attributed to relief of distress rather than enhancement of normal sensuous pleasure. A second type of explanation is based on deficient avoidance of the punishing effects of chronic, excessive drinking. This deficient avoidance is generally attributed to a self-destructive motive, which counteracts the normal, adaptive

behavior of seeking pleasure and avoiding distress.

4.1 DRINKING TO RELIEVE DISTRESS

Several applications of psychoanalytic theory are based on the disinhibitory effect of alcohol. Alcohol intoxication is pleasurable because it temporarily relieves conflicts and thereby relieves anxiety or frustration.

4.1.1 *Disinhibition of Homosexual Urges*

One of the influential early papers on alcoholism was by Karl Abraham (1908/1927). He emphasized the disinhibiting effect of alcohol, heightening sexual excitement and releasing suppressed perversions, such as homosexual feelings. He noted that the intoxicated person denies the impairment of sexual capabilities. He concluded that a greater tendency for and approval of active, disinhibited behavior in men than in women accounts for the preponderance of men who drink excessively and become alcoholics.

Abraham's discussion of alcoholism is consistent with Freud's published comments on the topic. Subsequent citations of Abraham's article have emphasized his brief comment on homosexual behavior as one of the sexual perversions released by the disinhibiting effects of alcohol. This elaborated on Freud's suggestion, in a letter to Fliess on 11 January 1897, that alcoholism is a substitution for a homosexual perversion (Strachey, vol.1, p.240; Masson, p.222). In 1910 Freud repeated this explanation in his account of the Schreber case (Strachey, vol.11, p.188). The theory of alcoholism as expressing a homosexual urge was summarized and elaborated many years later by Bergler (1944).

An article by Riggall (1923) added to the theory based on the disinhibitory effect of alcohol. Faulty psychosexual development causes the craving for oblivion, which is satisfied by intoxication. Men drink to overcome the repression of natural homosexuality, and women drink to bring out the male side of their bisexuality.

Blum (1966) distinguished three types of alcoholics in terms of fixation in three stages of development: oral, anal, phallic. Table 2 summarizes characteristics of alcoholics who are fixated at these different stages. In addition to Blum's typology, the three stages of development show the manifestation for each of the three components of the self. The last line of the table shows a classification by Blane (1968).

Fixation in the oral stage is expressed by impatience, intolerance of frustration, and denial of unpleasant reality. Riggall (1923) stated that the male homosexual identifies himself with his mother, seeks himself as his love object. Fixation in the anal stage is associated with aggressiveness, rebelliousness, and ambivalence. Fixation in the phallic stage is expressed by inability to express tenderness and sexuality toward the same person, and conflict with authority figures.

A more general extension of Abraham's principal theme focuses on the internal conflict that induces craving of the disinhibitory effect of alcohol. Release of the id impulses from the restraints of the superego and ego is unusually pleasurable when the conflict between id and superego is unusually

TABLE 2

**Alcoholics are classified by stage of fixation,
with characteristic psychiatric syndrome, manifestations
of self components, and typical reaction to alcohol**

	STAGE OF FIXATION*		
	Oral	Anal	Phallic
<i>Syndrome</i>	Depression	Neurosis	Sociopathy
<i>Id</i>	Passive	Self-destructive	Aggressive
<i>Superego</i>	Weak	Punitive	Denied
<i>Ego</i>	Ineffective	Divided	Allied with id
<i>Reaction to intoxication†</i>	Dependent	Dependent-independent	Counter-dependent

* After Blum (1966)

† After Blane (1968)

severe. Fenichel (1945) described the superego as the "part of the mind that is soluble in alcohol" (p. 379).

The pharmacological effect of alcohol on the central nervous system alleviates conflict. This helps to account for the pleasurable effect of intoxication. Barry (1977a) pointed out that the depressant action of alcohol decreases awareness of repression, thereby strengthening denial. A more general interpretation is that alcohol intoxication is pleasurable because it permits escape from perception of harsh reality; this was stated in one of the previously quoted passages by Freud (1930, in Strachey, vol.21, p.78).

4.1.2 Conflict over Dependency

Another theory regards conflict over dependency as the principal source of the unusual pleasurable effect of alcohol intoxication. Drinking liquor induces regression to the dependency that is a universal condition in the initial, oral stage.

One form of this theory is that alcoholics are predominantly assertive, denying their dependency desires. Their desires for dependency have been thwarted so that they unconsciously crave regression to the oral stage. Alcohol intoxication can satisfy this desire while also helping to maintain the denial from consciousness because of the impairment of mental functioning under the influence of alcohol.

This theory was proposed as an explanation for findings from a study on childhood characteristics of boys who subsequently became alcoholics (McCord et al., 1960). It has been applied to studies of ethnic and cultural groups by Snyder (1958) and by Bacon et al. (1965).

According to a subsequent version of this theory (Blane, 1968), a conflict over dependency can be manifested in three different ways: dependent, dependent-independent, and counter-dependent. The common feature is a focus on the conflict over dependency, in the oral stage of development. The bottom of Table 2 relates this classification of three types of alcoholics to fixation at the three stages of development. This implies that the behavioral manifestations of the alcoholic depend on the degree to which the individual also is fixated at the anal or phallic stages.

Some subsequent discussions of alcoholism are based on a conception of an infantile or disrupted personality. Khantzian (1982) emphasized the alcoholic's disturbance of sense of self. Krystal (1982) noted the prevalence of severe infantile traumatization and inhibition of the capacity for self-care.

4.1.3 *Psychotherapy*

Crowley (1939) and Blum and Blum (1967) have reviewed accounts of psychoanalytic therapy of alcoholics. Many writers have commented on the special difficulty of treating addictive behaviors, including alcoholism. The distress caused by some other neurotic symptoms is an incentive for therapy. For the alcoholic, however, resistance to therapy is increased by the pleasure and relief obtained from drinking. Relapse is likely to be precipitated by the distress that ensues from exposing defences and counteracting denial.

Prevention of drinking is generally regarded as necessary for effective therapy of alcoholics. Various deviations from the standard psychoanalytic technique have been described, with the therapy of alcoholics being generally more indulgent and also more directive. Friendly, extensive social contact with patients satisfies their oral dependency needs and counteracts their feelings of alienation and hostility. Weijl (1944) suggested that therapy is made more effective by reproducing elements of initiation and of rebirth because the onset of drinking is likewise often associated with initiation into a new stage of adolescence or adulthood that involves extensive changes in activities and self-concept.

Psychoanalytic therapy has often been characterized as most suitable for neurotic disorders. Neurotic symptoms cause misery, which is relieved by the therapy. The ego meanwhile is intact and able to cope with the stresses of increased self-knowledge. Alcoholism has some characteristics of a neurotic disorder, especially when anxiety and misery replace the pleasure and relief initially obtained from drinking. A predominance of neurotic rather than psychotic or psychopathic traits characterizes many alcoholics (Tamerin & Neumann, 1974).

4.2 SELF-DESTRUCTIVE MOTIVE

The disinhibitory effects of alcohol weaken the avoidance of its adverse consequences; the excessive drinking thus continues even after the initial pleasurable effects fade and are replaced by pain. Alcoholism might develop in people who are deficient in avoidance of the punishing consequences of excessive drinking and thus continue to seek the disinhibitory effect of intoxication.

Psychoanalytic theory identifies a self-destructive motive as the basis for

deficient avoidance of the punishing effect of excessive drinking. Anger and other destructive feelings can be directed against the parent incorporated into one's superego, possibly providing the basis for the self-destructive characteristic of alcoholics, emphasized by some commentators. An influential characterization of alcoholism as slow suicide was contributed by Knight (1937, 1938) and Menninger (1938).

A generalized self-destructive motive indicates fixation at the oral stage, when the undeveloped ego cannot resolve the conflict between the id and superego. This conflict induces the guilt and self-destructiveness that are prominent in some alcoholics (Barry, 1982a). Deprivations in the oral stage are associated with generalized feelings of worthlessness, resulting in depression and suicide. Goodwin (1973) summarized evidence for association of alcoholism with suicide.

The self-destructive behavior of alcoholism is also related to fixation in the anal stage of development. Freud's comments on dipsomania emphasize compulsion, ambivalence, and denial. The alcoholic often alternates between sobriety and drunkenness, between preserving and abandoning rigid self-control. This is consistent with fixation in the anal stage of development. Intoxication constitutes a rebellion against the anal traits of being orderly, precise, and clean. Many alcoholics are primarily neurotic rather than psychotic, alternating between self-indulgent intoxication and intense efforts at self-control (Tamerin & Neumann, 1974).

5. CHARACTERISTIC RESEARCH METHODS

Five principal methods have been used for developing and testing the psychoanalytic theory. The first three are clinical reports, studies of early development, and investigation of the behavior of alcoholics. These are based on observations or statistical analyses of individual alcoholics compared with nonalcoholics. The fourth method, cultural customs, places drinking and alcoholism in the context of the collective social norms and the worldwide scope of cultural variations. As a fifth method, psychoanalysts have used the powerful scientific technique of controlled experimental interventions.

The psychoanalytic theory of alcoholism includes concepts that are not directly observable, such as libidinal motives and repression. It is difficult to make crucial predictions about outcomes that would either confirm or refute the theory. For example, developmental experiences that appear to be similar could have opposite effects, either a direct response or reaction formation. Exaggerated pleasure from drinking, due to oral fixation, can cause either alcoholism or phobic avoidance of alcoholism. In spite of this formidable obstacle to research, diverse types of studies have provided substantial support for the psychoanalytic theory of alcoholism.

5.1 CLINICAL REPORTS

The original material consisted of clinical case studies by Freud and his

early followers. Their conclusions, while not provable, are based on extensive clinical experience. Psychoanalytic theory was initially developed from the free associations of patients, recorded and interpreted by the therapist. Repressed material is presumed to be made available to conscious awareness by this therapeutic technique, which encourages the patient to reveal feelings and memories with minimal interference from the psychoanalyst.

5.2 EARLY DEVELOPMENT

Two types of studies can obtain systematic information on the early development of alcoholics. One type, which relies on retrospective accounts by the patients, has the advantage of direct memory but the disadvantage of possible distortion of memory. The second type relies on accounts by relatives or other sources of information about the patient. More extensive studies can be based on objective information about a group of people. For example, birth order in the family is an attribute of the early environment that can be measured easily and accurately in a large sample.

5.3 BEHAVIOR OF ALCOHOLICS

Many inferences based on psychoanalytic theory have been derived from observations of behavior of alcoholics. The chronic, intense conflict thought to characterize the lives of many alcoholics is evident in the alternation between drinking and sobriety. A self-destructive motive is seen in the recurrent, self-damaging alcohol intake. The techniques include analysis of anecdotes, observations of individuals, and quantitative comparisons between a sample of alcoholics and nonalcoholics.

5.4 CULTURAL CUSTOMS

The scope of psychoanalytic theory is enlarged by applying it to the great variety of cultural customs. Psychoanalytic theory has been used to explain cultural variations in drinking customs. Systematic cross-cultural studies have been made possible by the availability of quantitative measures of alcohol use and of other variables in numerous societies in all the major inhabited regions of the world.

5.5 EXPERIMENTAL INTERVENTIONS

A uniquely convincing method for testing any theory is the controlled experiment. This method has seldom been applied to psychoanalytic theory because it is not feasible to manipulate experimentally the conditions of human development that are believed to influence susceptibility to alcoholism. Some scientists have tested brief interventions, comparing an experimental with a control procedure and using psychoanalytic theory to predict a therapeutic effect on alcoholics or amount of drinking by nonalcoholics in a laboratory environment. Another use of controlled experiments to test psychoanalytic theory is the development of a laboratory animal model of alcoholism, attempting to identify experiences in early development that reproduce the motivations and drinking behavior of human alcoholics.

6. BOUNDARIES OF THE THEORY

Psychoanalytic theory has important strengths. It attempts a comprehensive explanation of the development of all types of psychopathology, including alcoholism: the excessive drinking is explained by prior maladaptive learning. In the context of this explanation, vivid terms are used to identify three components of the self (id, superego, ego) and four stages of development (oral, anal, phallic, genital).

The theory also explains the pleasurable effects of drinking, recognizing the adaptive function of the temporary release from inhibitions, anxieties, and pressures of reality. In a context of social drinking, abstinence might be a neurotic, phobic response or it might be an adaptive method of warding off the temptation for self-destructive drinking.

The theory has serious limitations. The essential concepts are based on inference rather than directly observed actions or objects, and the components of the self and the stages of development are not clearly separated from each other. Alcoholism may be explained by fixations that are at one or more of several stages, that are caused either by deprivation or by excessive gratification, and that induce either the direct characteristic behavior or (due to reaction formation) the opposite behavior.

The abstract and complex nature of the psychoanalytic concepts leads to a further difficulty. Different theorists view stages of development and components of the self in different ways, using different terms; their elaborations of the theory create further obstacles to understanding and testing it.

It is difficult to prove or disprove the assumptions of the psychoanalytic theory. A systematic, logical analysis (Grunbaum, 1984) concludes that psychoanalytic theory is testable but that its concepts, such as motivated repression from conscious awareness, have not been convincingly verified. Alcoholism is not included, however, in this identification of topics that psychoanalytic theory fails to explain.

Psychoanalytic theory attempts to explain individual differences in terms of developmental experiences but not in terms of biological variations. Therefore, the theory does not explain the portion of individual differences caused by innate variations in physiology and temperament.

The psychoanalytic theory originated in late-19th-century European urban culture. Many human attributes presumed to be universal may be limited to that society. Stages of development, and their effects on the components of the self, might be different in other cultural environments. Alcohol also may have different meanings in different communities; for example, availability and price of liquor may account for excessive consumption of alcohol instead of other drugs, such as narcotics.

The psychoanalytic theory attempts to identify a specific pathological syndrome as the cause of alcoholism. This might not be possible, however, for such a variable and socially regulated behavior as alcohol consumption. Studies on personality traits of alcoholics cited by Barry (1974) identify different patterns of alcoholism, with no single characteristic applying to all

alcoholics, and some researchers have concluded that there is no one personality factor related to alcoholism.

7. RESEARCH RESULTS AND NEEDS

The research findings are discussed in relation to the characteristic research methods described in §5. The information contributed by each method is classified according to whether the alcoholism is attributable primarily to conflict at the phallic, anal, or oral stage of development.

7.1 CLINICAL REPORTS

An important basis for evaluating psychoanalytic theory is its success in helping alcoholic patients. A review by Voegtlin and Lemere (1942) indicated a preponderance of failure. Nevertheless, the theory may be valuable if it explains the failures and shows the way to more effective methods of treatment based on psychoanalytic principles. The alcoholic is resistant to the development of insight and to the minimal directiveness that are components of traditional psychoanalytic therapy. Krystal (1977) suggested that alcoholics do poorly in individual therapy because the therapist becomes the focus of their conflict between yearning for union with the maternal love object and aggressive death wishes. Countertransference, which is an important aspect of traditional psychoanalysis, is likely to drive the alcoholic back to drinking and away from the therapist.

Some writers have reported effective modifications of the standard therapeutic techniques. Knight (1938) recommended the combination of institutional treatment and psychoanalysis. Krystal (1977) suggested the use of a therapeutic team rather than a single therapist. Khantzian (1978) stated that the principal reason for the effectiveness of Alcoholics Anonymous is its emphasis on abstinence as the single most important goal. This program also compensates for the alcoholic's impairment of self-care.

Better therapeutic results have been obtained with the addition of directive therapy. A necessary part of the therapy is to prevent the patient from obtaining relief by drinking. The drug disulfiram can be helpful by precluding alcohol intake. Wallgren and Barry (1970) summarized studies showing effectiveness of this treatment. Etzioni and Remp (1973) summarized the uses and limitations of this treatment. The principal therapeutic value is in helping the patient to resist temptations to resume drinking, as is indicated by a study that found equal therapeutic effect in patients with a dose that was virtually a placebo, too low to induce the adverse reaction when alcohol was consumed (Fuller & Williford, 1980).

Some clinical observations cited by Barry (1974) have pointed out childhood experiences that predict later alcoholism. Alcoholics have been reported to have bad relationships with their parents or to have remained dependent too long. Such observations probably include valid insights, but there has not been adequate verification of their objective validity.

The initial clinical studies consisted of observations of cases. These reports have the disadvantage of being anecdotal accounts of a limited number of individual patients. The theoretical preconceptions might influence the therapist's observations or the patient's responses. On the other hand, such studies contribute a large amount of information that both the patient and therapist regard as valid.

The evidence on repressed homosexual strivings is mostly based on clinical reports. These strivings are assumed to be an intensified expression or repression of the bisexuality that is a normal component of males and females. Alcoholics have not been differentiated adequately from other types of patients or from normal adults in this respect.

Grunbaum (1984) has questioned the trustworthiness of clinical reports based on free associations by patients. The analyst may influence the course of free associations in undesired ways. This criticism expresses doubt about the conclusions that arise from clinical observations but does not refute them. Additional research techniques are obviously needed.

For psychoanalytic theory, the principal research need is development of more direct measures of the motives that are presumed to impel the person to drink addictively. Innovative interview techniques or projective tests might identify the multiple, conflicting motives that influence susceptibility to alcohol intoxication.

7.2 EARLY DEVELOPMENT

Some psychoanalytic case reports are based on early memories of patients. An example is in an article by Simmel (1948). "The parents of most alcoholics I have studied, father or mother or both, were usually emotionally immature, unstable persons. They permitted themselves indulgences and enjoyments which they prohibited their children." Consequently, "All my alcoholic patients had deeply seated hatreds for their mothers. This hatred is deeply repressed as an impulse to incorporate, to destroy by devouring the mother" (p. 20).

The accuracy of such memories is questionable. Therefore, an important alternative technique is to obtain objective information on early characteristics of alcoholics. In an early study of this type, McCord et al. (1960) identified the characteristics of boys who later displayed severe drinking problems. The findings led to their hypothesis about the role of denied dependency desires in the development of alcoholism. Jones (1971) reported a similar study on a small number of girls who became alcoholics.

Barry (1977b) summarized several studies indicating impulsiveness and deficient self-control among children and youths who subsequently became alcoholics. This is consistent with an explanation of alcoholism based on deficient ability to avoid the punishing consequences of excessive drinking.

Another technique has been to obtain objective information on the childhood characteristics of alcoholics. Birth order in the family has special value as a topic for study. This source of variation in the early environment is not pathogenic but could alter susceptibility or type of response to pathological influences. It is an objective measure, and accurate information can usually be

obtained on a large sample. Numerous studies of birth position of alcoholics were summarized by Blane and Barry (1973). Alcoholics are more likely to be last born in large families. The same tendency occurs for both sexes but is more consistently found in males.

Blane and Barry (1973) explained their finding on the basis of heightened conflict over dependency in the "baby of the family." An alternative possible interpretation is that early childhood development may be disturbed by disruption of the family, due to parental divorce, desertion, or death. If this occurs, the youngest child in the family is at the earliest age and hence presumably most vulnerable. This possible explanation of birth-order effects has been discussed by de Lint (1974) and by Blane and Barry (1974).

Subsequent research has revealed other attributes of birth order of alcoholics. A study by Conley (1980), with the desirable additional feature of a nonalcoholic control group, confirmed prior reports of excessive numbers of last-born children from large families. An additional finding was an excessive number of alcoholics who grew up without any siblings. Furthermore, Blane and Barry (1975) found that the second older sibling of alcoholics was more likely to be a sister than a brother. This tendency was subsequently reported in a small sample of presidents of the United States and their brothers (Barry, 1979).

These studies may identify early family experiences that increase vulnerability to alcoholism. Disturbance of masculine identification makes a boy more vulnerable to alcoholism. Such disturbance is more likely to develop in the last child (the "baby of the family"), in an only child, and in a boy who has a sister rather than brother in the influential position of second older sibling.

There is clearly a need to identify the other attributes of childhood family experiences that have the most consistent and important effects on subsequent personality. This task requires taking into account other variables that might be influential, for example by controlling for family size and socioeconomic status when analysing the effects of birth position.

Two contrasting types of early influences may protect the person against alcoholism. One of these influences is healthy, normal development that prevents the self-destructive behavior of excessive drinking; the other is a disturbance in development that induces alternative pathology. A possible example of both types of protective influence is the rarity of alcoholism among kings (Barry, 1979). The protective effect might be due to the strong sense of status that is developed, counteracting the feeling of alienation and disordered self-identity that characterize alcoholics. An additional factor is birth order, since kings are seldom last born. Some attributes of a royal childhood are not healthy, so that kings tend to develop alternative symptoms rather than to be free from psychopathology. Symptoms that are probably as frequent among kings as in the general population include paranoia and obsessive-compulsive neurosis.

7.3 BEHAVIOR OF ALCOHOLICS

No single technique is adequate for studying the behavior of alcoholics. Among the many techniques used, the principal types are clinical observa-

tions, projective tests, and statistical studies.

7.3.1 *Clinical Observations*

Many of the assumptions of the psychoanalytic theory are supported by observations of alcoholics. Anger, misery, and feelings of alienation are prevalent emotions in this group. Alcoholics direct their hostility primarily against themselves, but drunken violence against others often occurs.

There are some favorable attributes of alcoholics. They do not show the pervasive, generalized pathology seen in schizophrenia or severe depression. Many alcoholics are likable and socially adept, capable of functioning well though their egos may be weak and brittle. Furthermore, many alcoholics are able to abstain and enjoy good physical and mental health even after many years of excessive, damaging drinking.

The contradictory characteristics of alcoholics are expressed by their choice of alcohol intoxication as their symptom. The prevalent approval of social drinking makes it difficult for family and friends to deal with the transformation of this recreational activity into a destructive pattern of behavior. The differential effect of alcohol intoxication on nonalcoholics and alcoholics is portrayed in Table 1 (see §2.2).

Oral fixation in alcoholics is indicated by many expressions of dependent behavior when intoxicated, and the repeated consumption of a drug with incapacitating effects. For example, Simmel (1948) commented: "Instances are numerous of a man whose drinking begins either when his wife becomes pregnant or gives birth to his child" (p. 10). In a more extensive study, Browne (1965) reported a linkage of drinking to the nursing experience. Alcoholic men commonly increased their drinking shortly before and throughout the first year after the birth of their baby. Alcoholism in women often began shortly after childbirth. An observation that many alcoholics are married to nurses (Armstrong, 1958) was attributed to dependency needs, to seeking women who dominate and mother them. The widespread success of Alcoholics Anonymous may be attributable partly to oral dependence of the members on the group and its frequent meetings.

Fixation at the anal stage is evident in many alcoholics. Conflict over this stage is indicated by reports of alcoholic patients who are messy and troublesome when drunk but neat, clean, and docile when sober (Browne, 1965). The rebellious behavior of drinking excessively alternates with compulsive efforts to abstain. Anal traits are prominent in many alcoholics, including obsessive-compulsive neurosis (Tamerin & Neumann, 1974), anger, and ambivalence. Wurmser (1978, p.124) described anal traits of alcoholics in contrast with addicts to other drugs. In alcoholics "the main feelings denied appear to be guilt and loneliness, also in many shyness, shame, social isolation. The narcissistic gratification lies in the expression, not in the denial, of anger which had been so long suppressed or repressed." Wallgren and Barry (1970, vol.2, p.769) and Barry (1979) have commented on permissiveness and other attributes of Alcoholics Anonymous that counteract the antagonism and self-hatred of alcoholics. Simmel (1948) commented that through Alcoholics Anonymous "The alcoholic's psychopathological formula of destroy and be

destroyed is changed to save and be saved" (p. 31).

Fixation at the phallic stage is seen in disturbances of love relationships. Alcoholics tend to have unsatisfactory, antagonistic relationships with friends and lovers. Some alcoholics show evidence of homosexual desires that are repressed. Zwerling (1959), in a study of 46 alcoholic men, reported that 11 had adult homosexual experiences, most of which were attributed to intoxication. In addition, the author asserted, "A number indicated thinly veiled, latent homosexual impulses" (p.551).

Alcohol intoxication increases erotic desires while impairing capability. Vanderpool (1969) found that alcoholic men reported increased heterosexual feelings during an experimental session; the same effect occurs in normal drinkers (McClelland et al., 1972).

Interpretation of the behavior of alcoholics is difficult. It is frequently uncertain to what degree the behavior expresses prior personality characteristics and to what degree it is a consequence of the excessive drinking. The destructive consequences of excessive drinking account for much of the misery of alcoholism.

An additional research need is for better knowledge of the feelings associated with drinking. An obstacle to this quest is that intoxication itself obscures feelings. Nevertheless, useful information is contributed by observations of the emotions of alcoholics during drinking, such as studies by Mello and Mendelson (1970) and Mello (1972).

7.3.2 Projective Tests

Motivations of alcoholics have been explored with the use of projective tests that attempt to circumvent repression of the motives from conscious awareness. The validity of these tests is disputed, and the inferences are not specific to alcoholics. Moreover, projective tests are not completely successful in removing the barriers of repression from conscious awareness. Some of the findings, however, provide useful information about alcoholics.

Evidence for oral fixation of alcoholics is seen in responses to several diverse tests. With the popular Rorschach test, asking people to state the meanings they see in symmetrical ink blots, one study showed a large number of water perceptions by alcoholics (Wiener, 1956). This was interpreted as indicating an emphasis on oral or passive feelings. Evidence for oral fixation of alcoholics, resulting in a preference for soft, sweet liquid foods (Wolowitz, 1964), was supported by responses to a questionnaire on oral traits (Wolowitz & Barker, 1969). Oral fixation can be inferred from field dependence in tests that measure the degree to which the person adjusts passively to the environment rather than controlling it. Alcoholics showed an abnormally high degree of field dependence in a series of experiments with the rod and frame test (Karp & Konstadt, 1965; Karp et al., 1965a,b; Witkin et al., 1959).

Projective tests have also indicated fixation of alcoholics at the anal stage, expressing ambivalence between success and failure or between cooperative and resistant behavior. The Rorschach test responses by a sample of alcoholics led to the conclusion that the alcoholic's ambitions are rather high but the actual achievements are limited (Billig & Sullivan, 1943). Responses by

alcoholics to the Rorschach and other tests led to the interpretation that they do not recognize limitations or inadequacies in their personalities, and deliberately expose themselves to irritating or challenging stimuli instead of insulating themselves against them (Halpern, 1946).

Evidence for fixation of alcoholics at the phallic stage has been obtained by McClelland et al. (1972) from stories that are stimulated by a series of standardized pictures (Thematic Apperception Test). Alcohol intoxication induces fantasies of being powerful. This feeling denies the reality of the incapacitating effects of intoxication. The research was mostly done on young social drinkers, but the conclusions were generalized to alcoholics. McClelland et al. (1972) commented that many alcoholics marry domineering, managerial women. This pattern was viewed as a way to fulfil the fantasy of acquiring power.

Machover et al. (1959) identified measures that differentiated homosexual from heterosexual males in the Rorschach test, Machover Figure Drawing Test, and Masculinity-Femininity Scale of the MMPI. Partial resemblance to the homosexuals was found in the responses by a group of male alcoholics in remission but not by a group of male alcoholics who were unremitting but sober when they took the test. This finding supported the theory of latent homosexual feelings among male alcoholics, assuming that the projective tests measured these latent feelings more accurately in the remitted than unremitting alcoholics. Gibbins and Walters (1960) found that in a test of esthetic preferences, pictures with male rather than female symbolism were predominantly preferred by the homosexuals but not by the controls. The alcoholics, who apparently resembled the unremitting group of Machover et al. (1959), were intermediate between these two groups, being more similar to the controls according to the data presented but more similar to the homosexuals according to the statistical analyses and interpretations by the authors (Gibbins & Walters, 1960).

Rudie and McGaughran (1961) distinguished between two types of alcoholics (essential and reactive) by responses to the Rorschach and Sentence Completion tests. The essential alcoholic uses the simple, primitive defence mechanism of repression. He shows a psychopathic trend with more severe psychopathology and earlier onset of excessive drinking. The reactive alcoholic uses the more complex defence mechanism of reaction formation and shows a neurotic trend.

7.3.3 Statistical Studies

Useful information is obtained from quantitative comparisons of alcoholics with nonalcoholics with respect to selected variables. An advantage of these comparisons is that they can be applied to large numbers of people.

A systematic study by Vaillant (1983) obtained numerous objective measures of behavior and of the family and other environmental conditions during childhood development of a sample of urban males in order to identify the variables associated with subsequent alcohol abuse. Future alcoholics were more likely to be premorbidly antisocial and extraverted, in accordance with prior studies summarized by Barry (1982a). Reaction formation, a

concept contributed by psychoanalytic theory, was indicated by Vaillant's finding that lifelong abstainers shared many attributes of alcoholics and during childhood "seemed just as psychologically impaired as future alcohol abusers" (p.114). Barry (1968) pointed out cultural variations that indicated similar reaction formation among abstainers.

In analyses of statistical data on larger populations, some variables are conspicuously associated with vulnerability to alcoholism. One of these is the higher percentage of men than women who are alcoholics. This disproportion is consistent with a motivational explanation based on the fact that men are more strongly trained to deny their desires for dependency. Other possible explanations, however, include the larger number of opportunities for social drinking by men and the greater disapproval of drunkenness in women.

A high proportion of alcoholics have been married. Divorce is abnormally frequent, but sexual capability and interest are indicated by the behavior of marrying (Goodwin et al., 1973). Barry (1974) summarized studies on marital patterns. It is uncertain whether the high frequency of divorce is due primarily to predisposing characteristics or to the effect of the excessive drinking.

Studies of alcoholics have generally identified several types rather than a single alcoholic personality (Barry, 1974). This conclusion agrees with that of Cox in Chapter 5 in this volume. Various pathological attributes of personality have been shown to be frequent among alcoholics, however. All alcoholics by definition have in common a trait of persistent pathological behavior.

Statistical information does not adequately test the psychoanalytic theory that repressed homosexuality increases vulnerability to alcoholism (Abraham, 1927). Currently, overt homosexuality is associated with a high risk of alcoholism among males (Lohrenz et al., 1978) and females (Saghir & Robins, 1973). This evidence could be interpreted as supporting the psychoanalytic theory because many contemporary overt homosexuals would have repressed their homosexuality several decades ago. Other interpretations are possible, however, such as the prevalence in the gay community of making social contacts in bars.

The dependency theory has been supported by considerable evidence from statistical studies. According to studies cited by Barry (1982b), alcoholism is associated with conditions inducing alienation and anxiety, to which some people respond by regressing to dependency. These conditions include urban living and disruptive social changes.

Alcoholism is linked to depression. Chronic, excessive drinking has been found in a high proportion of people who commit suicide. Barry (1974, 1980) has summarized evidence for the association of alcoholism with other psychopathologies, including suicide, depression, manic-depressive illness, and self-destructive use of other drugs.

Another observation is poor impulse control by many male alcoholics (see Cox, Chapter 5). Sociopathy is one of the behavioral disorders associated with alcoholism (Barry, 1974).

Although alcoholism occurs more frequently in men, there are many women alcoholics. Studies of female alcoholics may help to identify the

protective attributes in this less susceptible population. Studies of psychiatric illnesses have indicated that alcoholism in women is often linked with depression. Evidence for conflict over femininity among female alcoholics (Jones, 1971; Wilsnack, 1973) suggests that unconflicted femininity is normally a protective factor.

7.3.4 *Progression of Alcoholism*

Repeated intoxications build up the habit of drinking excessively and weaken the defences against this self-destructive behavior. The physical impairment caused by chronic drinking can further weaken resistance against the pathological behavior pattern. Therefore, alcoholism tends to become progressively more severe.

Descriptions of alcoholics generally also indicate strong resistance. Variability in behavior is typical, patterned on the transitions between the sober and the intoxicated state. Changes in drinking behavior are presumed to depend on how severely the ego function is impaired.

Increasing age sometimes helps to alleviate the problem, possibly by counteracting impulsiveness or diminishing the need for relief. Many alcoholics become able to abstain from drinking. Some stop drinking after psychotherapy, or after joining Alcoholics Anonymous. Some stop drinking without any known therapeutic intervention.

There is a fierce controversy over whether alcoholics can develop a pattern of adaptive, controlled drinking. According to the psychoanalytic theory, pathological motives and habits can be remedied. Former alcoholics thereby might resume the pleasures of drinking without the compulsive craving or self-destructive excess.

Several studies indicate that some alcoholics subsequently maintain a pattern of controlled drinking without any apparent pathology. Wallgren and Barry (1970) summarized several such reports. Taylor et al. (1986) reviewed some subsequent studies. The rarity of this behavior is emphasized in both of these reviews and by most other commentators. The habit of pathological drinking is strong and tends to be elicited by the first drink. The disorders that induced the pathological craving may not be possible to cure completely. Most writers agree that abstinence is the preferable and only feasible method of control for most alcoholics, but vigorous debate continues (see, e.g., Stockwell, 1986, and *British Journal of Addiction*, 1987).

7.4 CULTURAL CUSTOMS

Some of the best support for the psychoanalytic theory of alcoholism has come from interpretations of studies of ethnic or cultural drinking customs. These include studies of national or ethnic groups and also statistical studies of customs in a world sample of tribal societies. Cultural variations in drinking customs have been summarized and interpreted by Barry (1976, 1982b).

An important source of variation among cultures is the degree to which the behavior of individuals is controlled internally or externally. This factor involves the distinction between social restraints and individual restraints. To what degree is the behavior of individuals controlled externally, by others? To

what degree are restraints internalized, so that people control themselves? Table 3 classifies societies on the basis of whether the internal and external controls over drinking are strong or weak. The external controls limit the frequency of alcohol intoxication. The internal controls limit the behavioral changes when drinking.

TABLE 3

**Societies are classified on the basis of
strong or weak internal controls over drinking
(left-hand and right-hand columns)
and strong or weak external controls over drinking
(upper and lower rows)**

INTERNAL RESTRAINTS

EXTERNAL RESTRAINTS		<i>Strong</i>	<i>Weak</i>
	<i>Strong</i>	Drinking lightly, on ceremonial occasions (Jews)	Drunken misbehavior is strongly punished (Scandinavians)
<i>Weak</i>	Frequent drinking without intoxication (Italians)	Frequent drunkenness; social disruption (Irish-Americans)	

A highly influential study, on drinking in Jewish culture (Snyder, 1958), was interpreted in accordance with psychoanalytic theory. Although alcohol use is well established, alcoholism is rare in that culture. This finding was explained by Jewish social solidarity, which minimizes conflicts over dependency that are assumed to underlie the craving for drunkenness.

Consistent with the conclusion by Snyder (1958) was a study of a large sample of societies by Bacon et al. (1965), showing a low degree of satisfaction of dependency needs of children in societies with a high frequency of drunkenness. Greater pressure on boys than girls to be self-reliant might account for the higher incidence of alcoholism in men than in women. This conclusion of Bacon et al., (1965) has been supplemented by further information and interpretations by Barry (1968, 1982b).

On Bacon et al.'s finding that dependency needs are prevalently denied or frustrated in societies with a high frequency of drunkenness, Barry (1968)

reported that the relationship was stronger in societies where drinking had been introduced by an alien culture than in societies with aboriginal drinking. It is generally maladaptive to become drunk in a society that does not encourage dependent behavior; the relationship can therefore be expected to be strongest in societies where drinking has recently been introduced, so that the cultural customs have not yet adjusted by providing more support for drunken people or by decreasing the need of drunken people for help.

A limitation on studies of alcoholism in tribal societies is that the available measure is generally drunkenness but not alcoholism. In most such societies, alcoholic beverages are not sufficiently available to permit frequent drunkenness. It is a reasonable inference, however, that in societies where alcohol is sporadically available, severe drunkenness expresses the same motivations as does chronic alcoholism in societies where many people have frequent access to liquor.

In societies where liquor is frequently available, different patterns of drinking may be expected, depending on whether personal and social restraints are weak or strong. Table 3 shows a model for these cultural conditions, with examples of some cultural differences. Of course, the typical drinking pattern for each type of culture applies only to some of the members, not uniformly to all.

7.5 EXPERIMENTAL INTERVENTIONS

A report of a therapeutic effect is based on the psychoanalytic theory of repression of infantile cravings. Schurtman et al. (1982) found evidence that excessive drinking is motivated by feelings of alienation from a maternal figure. Alcoholics were better able to abstain if they were presented with a message "Mommy and I are one" by tachistoscopic projection, so briefly that the patient was not consciously aware of the message. Silverman et al. (1982) have reported a series of studies indicating a therapeutic effect of this subliminal message on various other pathological conditions, such as obesity.

This effect of a briefly presented message suggests a possible effective test of psychoanalytic theory. The response to subliminal perception presumably evokes repressed feelings. This supposition is consistent with the assumption that alcoholism is motivated by dependency needs, which during sobriety are counteracted by feelings of alienation and hostility. Evocation of the repressed dependency motive may provide alternative gratification, thus counteracting the craving for liquor.

This interpretation is not convincingly confirmed by this one study. The experiment was limited to a single therapeutic message, compared to one control message, "People are walking." The objective of psychoanalytic technique is to strengthen the ego by increasing conscious awareness of the conflict between id and superego motives. The therapeutic procedure with the subliminal message shows more similarity to hypnotic suggestion than to traditional psychoanalysis.

One of the prominent effects of alcohol is to decrease self-awareness. Wallgren and Barry (1970) and Barry (1973) reported that people performing a task are usually not aware of the impairment caused by alcohol. In accordance

with this effect, Hull (1981) suggested that alcohol intoxication may be motivated by the desire to block adverse self-appraisal.

In a review of several studies, Hull (1981) concluded that experimentally manipulated failure feedback induced more drinking by people who were highly self-conscious according to their responses to a questionnaire. In terms of psychoanalytic theory, alcohol intoxication provides more effective relief to those whose conflict between the id and the punitive superego induces low self-evaluation. A limitation to this interpretation of Hull's findings, however, is that the motives of alcoholics were inferred from experiments on nonalcoholics.

Animal models of alcoholism have been attempted, based on psychoanalytic theory. Conger (1951) and Barry and Miller (1962) demonstrated in rats that alcohol diminished avoidance of a painful shock associated with food. This finding was interpreted as indicating that alcohol alleviated fear and anxiety. The disinhibited behavior is adaptive if the pain is not forthcoming or if the benefit of the food exceeds the danger and stress of pain. Contrary to this interpretation, Cappell and Herman (1972) criticized the empirical support for the assertion that alcohol decreases anxiety. Subsequently, another type of therapeutic effect of alcohol was reported by Kraemer et al. (1981). A moderate dose of alcohol diminished the despair response of rhesus monkeys separated from the group of monkeys.

There have been many attempts to establish laboratory animal models for excessive intake of alcohol by humans. The animal models, however, have not been convincing reproductions of human situations. Laboratory animals have not developed the chronic, compulsive alcohol intake that characterizes alcoholics (Wallgren & Barry, 1970). A successful animal model for alcoholism may be developed if the animals can reproduce the human behavior of drinking to obtain relief. An example of progress toward this goal may be a report by Kraemer and McKinney (1985) that juvenile rhesus monkeys drank more alcohol if they were intermittently separated and united than if they were continuously isolated. They drank more during the isolation than reunion phases. For psychoanalytic theory, a more important contribution would be to reproduce in laboratory animals the human stages of development, fixation, and disturbance among components of the self, described by the theory.

8. PRACTICAL IMPLICATIONS

Psychoanalytic theory is oriented toward psychotherapy. Therefore, its practical implications have emphasized recommendations for therapeutic techniques. Weijl (1944) and Blum and Blum (1967) give advice on techniques for psychoanalytic therapy.

Widespread applications of psychoanalytic theory to alcoholism have been prevented by the general lack of success of psychoanalysis as a therapeutic technique for alcoholics. Nevertheless, psychoanalytic theory has led to proposals for therapy more suitable to alcoholics. Psychoanalytic theorists

have recommended the inclusion of Alcoholics Anonymous in most therapy programs. AA provides for many alcoholics an effective support group after detoxication.

The psychoanalytic theory of alcoholism is more a historical than a contemporary influence on therapeutic practice. Most descriptions of alcoholics and therapeutic programs are not explicitly based on this theory and do not use its language. More widespread acceptance of the theory would require stronger evidence, especially from experimental studies. Such studies would need to identify specific sources of alcoholism in fixations at stages of development and in disturbances of the components of the self.

The psychoanalytic theory has contributed more to understanding and therapy for alcoholism than is acknowledged in the literature. Social policy and child rearing have been influenced by its models of healthy and pathological development. In our contemporary society, there is increased emphasis on providing love and security for infants and children. More awareness is shown of the need to minimize antagonism and alienation during early development (Ginott, 1965).

Psychoanalytic theory emphasizes the importance of conflicting and diverse motives as causes of behavior. This attribute of the theory is especially helpful for understanding the ambivalent, erratic behavior of many alcoholics, alternating between sobriety and intoxication, between self-preservation and self-destruction. The psychoanalytic description of alcoholism as a slow suicide has the useful effect of emphasizing the seriousness of the pathological behavior. It contributes to the disease concept of alcoholism by emphasizing the need to halt a dangerous, life-threatening behavior pattern. It counteracts the popular myth of the comic, happy drunk.

9. RELATIONS TO OTHER THEORIES

Psychoanalytic theory attempts a comprehensive explanation of normal and pathological learning. Its purpose is commendable but not fulfilled. It is not generally accepted as a valid, general theory. Many people dispute its assumptions and propose alternative theories, such as those summarized in other chapters in this volume.

The psychoanalytic theory itself can identify sources of resistance, motivating rejection of psychoanalytic theory and advocacy of rival or supplemental theories. Such resistance can arise from motivations of the critic rather than the quality of competing theories.

9.1 RESISTANCES AGAINST THE THEORY

Our evaluation of a theory is a rational behavior. We use verbal skills and integrative ego functions to perceive and understand. It is difficult, therefore, to reconcile this logical function with the psychoanalytic theory, which emphasizes irrational, nonverbal behaviors. The conflict with our logical thinking impedes acceptance of each of the three principal elements of psychoanalytic theory.

Seeking sensuous satisfaction. Our contemporary society encourages people to value the rationality of ego functions. We are encouraged to believe that our motives are civilized, such as for accomplishment or improving human society. We deny the urgent, immediate craving for pleasure and our compulsion to obtain it. This repression enables alcoholics to deny that they have lost control over their behavior while they drink compulsively and self-destructively.

Conflict among components of self. The ego is the locus of self-awareness. Our desire for logical rationality induces the ego to deny the existence of concurrent, conflicting id and superego motives. For example, we do not recognize our simultaneous feelings of love and hate toward people who are close to us, including our parents, spouse, and children. We are generally aware of only one component of our ambivalent feelings, complete affection at some times and complete antagonism at other times. We alternate between consciousness of the desire to do our duty and consciousness of our rebellion against duty. The alcoholic alternates between sobriety and drunkenness.

Fixation in the infantile past. People wish to regard themselves as mature, and the persistence of childish traits is therefore denied. The nature of the earlier stages of development is altered to hide their obnoxious attributes. The oral stage of infancy is regarded as contented and loving instead of greedy and selfish. The anal stage is regarded as obedient and controlled instead of resistant and hostile. The phallic stage is regarded as sociable and sexy instead of exploitative and destructive. Instead of altering these terms, many people reject them completely because they describe physical cravings associated with immature stages of development.

Role of alcohol intoxication. The effects of alcohol contribute to resistance against the psychoanalytic theory. The ego function of logical awareness is impaired by alcohol intoxication (Barry, 1977a). This effect enables drinking to satisfy contradictory needs, such as for dependence (Blane, 1968) and fantasies of being powerful (McClelland et al., 1972). Alcoholism adds further sources of resistance. Denial of reality is a prominent characteristic of alcoholics. The denials may be applied to the urgent craving for sensuous satisfaction, or to the conflicting id and superego motives that impel the self-destructive behavior, or to the infantile learning that underlies the excessive drinking.

9.2 RIVAL THEORIES

Some theories are competitors, offering different explanations of the same type as the psychoanalytic theory. The advocates of these theories therefore try to contradict assumptions of the psychoanalytic theory. Two of Freud's prominent early followers were Alfred Adler and Carl Gustav Jung, both of whom developed theoretical disagreements with Freud and founded rival theories of mental illness. They and their followers have paid less attention to alcoholism than does the Freudian psychoanalytic theory. The comments about alcoholism in these rival theories are useful, however, to indicate some characteristics of the psychoanalytic theory.

Adler emphasized social learning rather than biological stages of development (Ansbacher & Ansbacher, 1956). He suggested that alcoholics usually

were pampered in childhood. They were dependent on others, usually exploiting the mother, and were later deficient in the ability to renounce the immediate gratification offered by alcohol intoxication.

Birth position in the family is an important variable according to Adler. Barry and Blane (1977) explained the relationship of birth position to alcoholism in terms of Adler's theory. The last child in a large family is more likely to become alcoholic (Blane & Barry, 1973). Pampering of this child by the parents and older siblings is indicated by the frequent designation "baby of the family." The inferiority complex gives rise to a need to compensate by feeling powerful; alcohol intoxication induces that needed feeling.

Psychotherapists following in the tradition of Adler have emphasized social learning and social relationships. The alcoholic is described as feeling alienated from other people and seeking comfort by drinking. The self-destructive behavior of chronic, excessive drinking thus is regarded as a consequence of the social alienation. Psychoanalytic theory regards the excessive drinking as an expression of a deep conflict, leading to this self-destructive behavior. The social alienation is a further symptom of the conflict and self-destructive behavior.

Jung made remarkably few comments about the cause or treatment of alcoholism, although reporting a high incidence of alcoholism among some categories of patients. He pointed out with disapproval that alcohol intoxication impairs mental functioning, but he appeared to regard it as a defect that is not accessible to treatment. He expressed more interest in the ritual symbolism of wine than in the psychopathology of excessive consumption. Jacobi (1967) stated that the goal of Jungian analysis, comprehensive development of the personality, may be inhibited by alcoholism or other cravings (p.17). Another passage pointed out the difficulty of curing alcoholism:

...an alcoholic, in order to be cured, must not only be conscious of his tendency or compulsion to drink — which many of them deny — but must also discover the deeper reasons that have induced his craving. These reasons are always shadow qualities which he cannot accept, which he flees from in order to rid himself of the pangs of conscience their recognition would entail. (p.40)

Conditioning theory and social learning theories attempt to explain alcoholism by prior learning, in common with psychoanalytic theory. Instead of the early developmental learning, however, these theories are based on learning more directly associated with the drinking. Some learning theories explain alcoholism as persistence of a response that relieves distress. Vogel-Sprott (1972) suggested that alcoholics are deficient in ability to learn to avoid the aversive effects of drinking.

These mechanisms for learning do not seem to be adequate explanations. More powerful influences seem necessary to account for the severely and persistently self-destructive behavior of excessive drinking.

9.3 SUPPLEMENTAL THEORIES

Some theories do not compete with psychoanalytic theory but instead broaden the explanation to take into account factors ignored by psychoanalytic theory. These include theories based on biological and cultural variations.

The biological theory, that some people are constitutionally susceptible to alcoholism, is compatible with psychoanalytic theory. The same pathological effect, such as alcoholism, may be explainable either on a biological or psychoanalytic level.

An example is disordered sexual orientation. An experiment on male rats demonstrated an effect of the early experience of suckling a mother whose nipples had a distinctive lemon odor (Fillion & Blass, 1986). In adulthood, the same lemon odor was necessary to elicit full mating behavior. This finding is explainable both by the psychoanalytic theory of the mother as the original model for heterosexual love and by the biological mechanism of imprinting. This example may be especially relevant to alcoholism because sexual deviations, such as homosexual desires, have been associated with alcoholism.

The pathological behavior of alcoholism may be due to a combination of pathological developmental experiences, explained by psychoanalytic theory, and constitutional vulnerability, explained by biological theories. A conspicuous attribute of alcoholism is that it occurs in only a minority of those who are at risk. This fact is consistent with the epidemiology of various types of psychiatric and physical diseases for which the occurrence requires a combination of constitutional vulnerability and precipitating conditions.

A combination of influences reported by Barry (1982a) was based on information on genetic and social variables provided by Donald W. Goodwin, expanding a published report (Goodwin et al., 1973). A high proportion of divorce among young men (33%) occurred only with the combination of two attributes: the young men showed evidence for alcoholism and had a biological parent who was alcoholic. The biological theories thus have the potential to fill a gap in the psychoanalytic theory.

The biological theories have developed more recently, when there has been more information about the pharmacological effects of alcohol and the physiological responses to this drug. The psychoanalytic theory constitutes a needed extension beyond the biological theories. Categories of people who are at risk of developing alcoholism have been identified, such as Irish-Americans or children of alcoholics. The majority of them do not become alcoholics, however. It is likely that alcoholism requires a combination of constitutional vulnerability, explainable by biological theories, and pathological developmental or environmental experiences, explainable by psychoanalytic theory.

Sociological and anthropological theories have the limitations inherent in describing individual behavior in terms of groups of people. Cultural customs influence the opportunity or temptation for excessive drinking. Determinants of alcoholism, such as frustration of dependency needs, have been inferred from characteristics of societies where drunkenness is frequent. Alcoholism is pathological behavior of a minority of individuals, however, rather than the prevalent custom in any society. Any society where most people are chronic alcoholics would not be expected to survive long. In accordance with that expectation, no such society has been observed. A complete theory of alcoholism therefore must explain individual pathological behavior, as do the psychoanalytic and biological theories.

10. CONCLUDING COMMENTS

Psychoanalytic theory has attributes that make it uniquely suitable for explaining alcoholism. The theory's explanations of pathological behavior can easily be adapted to the self-destructive behavior of chronic excessive drinking. The emphasis on seeking pleasure as a motive for behavior is consistent with the voluntary behavior of alcohol consumption. The variety in types of alcoholics can be explained by interactions between the successive stages of development (oral, anal, phallic, genital) and the different components of the self (id, superego, ego). The psychoanalytic theory has the advantage of attempting a comprehensive explanation of pathological behavior. The theory thus can account for pervasive and diverse maladaptive behaviors of alcoholics. The complex, interrelated developmental influences, in addition to biological differences, can explain why alcoholism does not develop in all people who are exposed to what appear to be the same pathological experiences.

It is understandable that many people dislike the psychoanalytic theory. Its emphasis on irrational motives and internal conflicts is contrary to the social training of humans to be logical and consistent. The theory is based on universal stages of development and components of the self that are theoretical concepts, not directly observable. No single, unified cause of alcoholism is suggested.

Biological factors are not adequately included in the psychoanalytic theory of alcoholism. The physiological effects of alcohol are taken into account only by the general description of alcohol as a toxic agent with disinhibitory effects, but biological variations among drinkers are ignored.

Psychoanalytic theory was developed when knowledge about effects of alcohol was meagre. This theory has not been modified to take into account subsequent information about biological aspects of alcohol. A synthesis of the new biological information with the psychoanalytic theory is needed to expand our understanding of alcoholism.

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5. PERSONALITY THEORY

W. Miles Cox

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1. PRINCIPAL ELEMENTS OF THE THEORY

1.1 DEFINING PERSONALITY

In order to delineate the principal elements of personality theory with respect to alcoholism, we must first of all understand what is meant by the term "personality."

As used in everyday conversation, personality usually refers to a person's social skills in interacting with other people. One person might be described as having a "good" personality or "more" personality than another person. As used by psychologists, on the other hand, personality usually refers to the totality of a person's behavioral characteristics. No attempt is made to evaluate these characteristics as desirable or undesirable.

Psychologists, nevertheless, do not have a precise definition of personality upon which they generally agree. The term has been defined in different ways by different authors (Feshbach & Weiner, 1984, p.12; Levy, 1970, pp.8-10). For purposes of this chapter, we will define personality as the organized pattern of a person's behavioral and emotional characteristics that distinguishes him or her from other people.

Psychologists use personality as a construct to help describe, organize, and understand people's behavior. Thus, personality refers to people's characteristic manner of responding across time and in a variety of situations. In characterizing people's manner of responding, we are concerned both with the consistency of their behavior from one occasion to another (within-person variations) and with how their manner of responding differs from that of other people (between-person variations).

People's personalities are thought to come from within themselves—that is, to have an internal locus of causation (Levy, 1970). Nevertheless, we know that personality originates from a variety of converging influences, including biological, psychological, environmental, and sociocultural ones. Klinger (1977, 1983), in fact, views personality as the branch of psychology that accounts for "how the parts [subsystems] of a person come together to produce total behavioral outcomes" (1983, p.30).

1.2 SITUATIONAL SPECIFICITY OF PERSONALITY

The patterns of behavior that people display depend on the situation in which they find themselves. For example, a person might be shy and reserved in one situation but talkative and outgoing in another.

This point of view has been emphasized by situationist theorists (e.g., Mischel, 1968, 1979). They argue that since observed behavior always depends on the situation in which it occurs, it is meaningless to view personality in terms of stable traits. A stance that is intermediate between the trait and situationist approaches has been taken by interactionist theorists (e.g., Endler & Magnusson, 1976) who argue that observed behavior results from the interaction between stable personality traits and the situation in which the behavior occurs.

1.3 TWO TRADITIONS FOR STUDYING PERSONALITY

There have been two major traditions for studying personality: the intrapsychic tradition and the differential tradition. Gordon Allport's definition of personality exemplifies the intrapsychic tradition: "the dynamic organization *within the individual* of those psychophysical systems that determine his characteristic behavior and thought" (Allport, 1937, p.28; italics added). The differential tradition is represented by Henry Murray's term personology: "the branch of psychology which principally concerns itself with the study of human lives and the factors that influence their course, which investigates *individual differences*" (Murray, 1938, p.4; italics added).

The intrapsychic tradition is represented by both the psychodynamic and experimental approaches to understanding personality. Both approaches start from the compelling phenomenological position that behavior is motivated by identifiable intrapsychic processes such as needs, motives, and drives. If, therefore, we examine differences in what people tell us about the structure of these processes within themselves, then we will better understand and predict behavior. The differential tradition, on the other hand, urges an empirical approach, free of preconceptions, on the basis of which a description of personality and its important dimensions can presumably be inductively derived. Both approaches have advantages and disadvantages. Whereas the intrapsychic approach may start on the basis of unvalidated constructs, a strictly empirical approach has no non-arbitrary starting point when trying to measure personality.

1.4 PERSONALITY THEORY AND ALCOHOLISM

There is no single personality theory of alcoholism. Instead, personality

theory vis-à-vis alcoholism refers to a point of view regarding how alcoholics' personalities are related to the development and maintenance of their alcoholism.

1.4.1 *Intrapsychic and Differential Approaches*

Psychologists working within the intrapsychic tradition have sought to identify alcoholics' needs, drives, and motives that would account for their involvement with alcohol. Psychologists working within the differential tradition have sought to identify the personality characteristics that distinguish alcoholics from nonalcoholics.

The intrapsychic approach to studying alcoholism was originally exemplified by psychoanalysts, who sought to explain alcoholics' unconscious motivations for using alcohol. More recently, the research of McClelland and his associates (McClelland et al., 1972) provides an apt illustration of using the intrapsychic approach to investigate alcohol problems. This research group discovered that males who drink heavily have strong needs to feel powerful and that drinking alcohol helps them to fulfil these needs. By contrast, the differential approach to studying alcoholism has been exemplified by the administration of personality tests to groups of alcoholics and nonalcoholics in order to identify the personality characteristics that distinguish the two groups.

Both the intrapsychic and differential approaches have proved valuable. Consequently, neither approach should be rejected in approaching alcoholism from the standpoint of personality theory. Although the two traditions represent very different approaches to research, they are not mutually exclusive. Moreover, the ultimate goal of the two approaches is the same: to identify the personality characteristics of alcoholics that would explain why they abuse alcohol.

1.4.2 *Topics for Investigation*

Some of alcoholics' distinctive personality characteristics are assumed to precede and others to result from their excessive use of alcohol. Consequently, there actually are three kinds of investigation that personality theorists tend to undertake. These aim to identify: (1) the personality antecedents of alcoholism, (2) the personality characteristics of alcoholics, and (3) the effects of alcohol use and abuse on personality.

Investigators hope that by delineating the personality characteristics of alcoholics, they will better be able to explain why alcoholics drink excessively. In arriving at such an explanation, however, it is important to assess which personality characteristics were apparent before the onset of alcoholism and which ones are a consequence of the alcoholics' involvement with alcohol. Isolating the personality precursors of alcoholism would help to elucidate future alcoholics' initial motivation for using alcohol (etiological factors), which may be quite different from their motivation for continued excessive use. With regard to the effects of excessive alcohol use on personality, it is important to distinguish between the direct effects of ethanol and the indirect consequences of leading an alcoholic lifestyle. Although in theory the separation of the personality antecedents and consequences of alcoholism is a

valuable goal to pursue, there are some practical difficulties involved in achieving it, which we will review later.

2. HISTORICAL DEVELOPMENT OF THE THEORY

The role accorded to personality in the etiology of alcoholism has undergone considerable change during the present century. At various times, personality has been seen (1) not to be a contributor to alcoholism, (2) to be the exclusive cause of alcoholism, and (3) to occupy a role in the etiology of alcoholism as important as biological, other psychological, and environmental influences.

2.1 EARLY HISTORY OF THE THEORY

The first suggestion that alcoholism is related to personality seems to have been made by psychoanalyst Karl Abraham (1908/1927), who proposed that alcoholism is a manifestation or symptom of alcoholics' difficulties with their sexuality. During the early part of the century, a number of other Freudian writers (see Blum, 1966; Fenichel, 1945; Jelliffe, 1917) expressed similar points of view. In spite of the contentions of these early writers, however, historically personality theorists have given very little attention to alcoholism. For example, even though opinions about the etiology of alcoholism were eventually strongly influenced by psychoanalysis, we see only passing mention of alcoholism in the 24 volumes of *The Standard Edition of the Complete Psychological Works of Sigmund Freud* (Freud, 1953/1966). The failure of personality theorists to consider alcoholism probably reflects the generally negative attitude toward alcoholics that has prevailed among both professionals and the general public to this day. That is, alcoholics have been viewed as persons unworthy of help, and until recently alcoholism was not considered an attractive topic for scientific inquiry.

During the early part of this century, alcoholism was usually attributed to the moral weakness of alcoholics or to the "demonic" properties of alcohol itself. Alcoholics, it was believed, were persons who lacked the moral fortitude to resist alcohol, and alcohol problems could be eliminated only by the removal of alcohol from society. This point of view gained political force and culminated in the United States with the passage of the Eighteenth Amendment to the Constitution which prohibited the manufacture, sale, and transportation of intoxicating liquors and which became effective in 1920. The attempt to prohibit alcohol proved, of course, to be a disastrous political failure, and Prohibition was repealed by the Nineteenth Amendment which became effective in 1933. Similar legal prohibitions against alcohol that were later repealed occurred in Canada and the European countries.

2.2 POST-PROHIBITION ERA

Following the repeal of Prohibition, alternative causes for alcoholism were considered, and attitudes toward alcoholics began to change. One impetus for the change was psychoanalysis. Psychoanalysts appear increas-

ingly to have accepted alcoholics into treatment, and they published articles describing their clinical impressions about the etiology of alcoholism (e.g., Chassell, 1938; Daniels, 1933; Knight, 1936, 1937, 1938; K.A. Menninger, 1938; W.C. Menninger, 1938). Like the earlier writers, psychoanalysts during the 1930s often attributed alcoholism to sexual difficulties or to various other forms of psychopathology stemming from early childhood experiences.

A second impetus for change was Alcoholics Anonymous. This self-help organization for alcoholics, which was founded in 1935, subscribed to the disease concept of alcoholism (i.e., that alcoholism is a disease rather than a moral weakness) as its guiding philosophy. The idea that alcoholism is a disease actually had been proposed much earlier (Rush, 1814/1943), but its promotion by Alcoholics Anonymous gave it widespread popular appeal. At the same time, the disease concept gained professional respectability because of the close association between Alcoholics Anonymous and E.M. Jellinek, a scientist who later published his views in *The Disease Concept of Alcoholism* (Jellinek, 1960). According to Alcoholics Anonymous, alcoholics must undergo fundamental changes in their personality and lifestyle in order to remain totally abstinent from alcohol, thereby keeping their disease in remission (Thoreson & Budd, 1987).

2.3 THE "ALCOHOLIC PERSONALITY"

During the 1940s and 1950s, there were various references to the concept of the "alcoholic personality" (Landis, 1945; Levy, 1958; Machover & Puzzo, 1959), which carried with it the idea that alcoholics have a unique personality structure that is both necessary and sufficient for alcoholism to occur. Thus, in contrast to the dominant view at the beginning of the century that alcoholism is unrelated to personality, the "alcoholic personality" implied that personality was the exclusive cause of alcoholism. The concept of an alcoholic personality was undoubtedly fostered by the important role that psychoanalysts assigned to personality in the etiology of alcoholism. However, the concept was also consistent with the views of Alcoholics Anonymous. Members of Alcoholics Anonymous sought to find a fundamental cause for the disease with which they were afflicted, and it was entirely reasonable to assign the cause to a disordered personality.

Concurrent with speculation about the existence of an "alcoholic personality" were efforts to specify its nature by administering psychological tests to alcoholics. These tests included projective tests such as the Rorschach Inkblot Test (Machover & Puzzo, 1959), objective tests such as the Minnesota Multiphasic Personality Inventory (MMPI; Hewitt, 1943), and various tests of perception/personality interactions (Witkin, Karp, & Goodenough, 1959). (See §3 and §5 for a description of these various tests.) From these efforts to measure the alcoholic personality, agreement was never reached about the exact nature of the alcoholic personality or, for that matter, whether it existed at all (Armstrong, 1958; Seliger & Rosenberg, 1941; Sutherland, Schroeder, & Tordella, 1950; Syme, 1957).

In spite of the unsuccessful attempts to identify the alcoholic personality, the practice of administering personality tests to alcoholics in an attempt to

identify their distinctive personality characteristics has continued unabated to the present day. However, the search for the alcoholic personality has been gradually replaced by the search for various personality subtypes that help to account for the alcoholism of different persons. Although the heterogeneity of alcoholics has long been recognized (Knight, 1937; Levine & Zigler, 1973; Rudie & McGaughran, 1961), we will later see that most of the attempts to identify subtypes of alcoholic personalities have occurred relatively recently.

2.4 EARLY CROSS-CULTURAL AND LABORATORY RESEARCH

Despite the lack of agreement that psychometric tests provided about the nature of the alcoholic personality, evidence from cross-cultural studies of drinking behavior and from the experimental psychology laboratory bolstered the view that one aspect of personality (*viz.*, one's affective tendency) is indeed a contributor to alcoholism. Specifically, cross-cultural studies (e.g., Horton, 1945) were interpreted as demonstrating that societies that drink heavily have high levels of "subsistence anxiety," and laboratory studies with animals (e.g., Conger, 1951; Masserman & Yum, 1946) suggested that alcohol "reduces tension" and that animals will drink alcohol for its tension-reducing effects.

From these two sources of evidence, the tension-reducing hypothesis of alcohol consumption was later formulated (Conger, 1956). It holds that alcohol reduces tension and that organisms drink alcohol in order to reduce their tension. Moreover, from this hypothesis we would expect alcoholics to have high levels of tension (*i.e.*, anxiety) that they attempt to alleviate by drinking alcohol. The tension-reduction hypothesis strongly influenced subsequent research, which sought (1) to distinguish alcoholics from nonalcoholics in terms of their chronic level of anxiety (see Cox, 1979, 1985) and (2) to determine the specific effects that alcohol has on the anxiety of alcoholics (see Langenbucher & Nathan, 1983, *in press*).

2.5 CURRENT STATUS OF THE THEORY

Despite the longstanding interest in the relationship between personality and alcoholism, significant strides toward understanding that relationship have occurred only recently (Cox, 1983). Since 1970, there has been a proliferation of scientific research on alcoholism, and a substantial portion of this research has dealt with the personality correlates of alcoholism. The research findings can be interpreted as leaving no doubt that personality is, in fact, a significant contributor to the onset and development of alcoholism. However, in contrast to the former view that a unique personality structure is the exclusive cause of alcoholism, the current view is that various personality dimensions interact with various biological, environmental, and other psychological variables to produce the complex phenomenon of alcoholism.

3. CHARACTERISTIC RESEARCH METHODS

There are two goals for the present section. First, we will discuss the types of personality tests that are used in research studies on alcoholism.

Second, we will describe the methodology that is used in studies of alcoholism from the perspective of personality theory.

3.1 PERSONALITY TESTS

Investigators who follow the intrapsychic approach and those who follow the differential approach both utilize personality tests as tools for studying the relationship between personality and alcoholism. Traditionally, personality tests have been classified into one of two broad categories: projective and objective.

3.1.1 *Projective Tests*

Projective tests of personality were first developed by psychoanalysts to assess their patients' unconscious personality structure, especially their unconscious needs and motives. Subsequently, highly quantitative, nonintuitive (and even computerized) systems for scoring and interpreting projective tests (e.g., Exner, 1974; Piotrowski, 1964) have been developed. Nevertheless, in actual practice, the interpretation of the results of projective tests, unlike that of objective tests, has often involved clinical intuition and subjectivity (Exner, 1976, p.67).

With all projective tests, there is a discrepancy between the stimuli provided to the test taker and the material that he or she is required to produce in order to complete the test. Since the required material cannot come either from the test stimuli or the subject's memory, the material must be projected from within the subject himself or herself. By projecting in this manner, the subject unwittingly reveals information about his or her own psyche.

Even though all projective tests involve such a discrepancy, the degree of discrepancy varies widely from test to test. For example, of the two most widely used projective tests, the Rorschach Inkblot Test and the Thematic Apperception Test, the discrepancy is great in the former, whereas in the latter it is less so.

Rorschach Inkblot Test. The Rorschach Inkblot Test (developed by Hermann Rorschach in 1921) consists of a series of 10 white cardboard plates, each of which has a symmetrical inkblot on it. Five of the inkblots are black and grey; two are black, grey, and red; and the remaining three are multicolored. The person being administered the test is handed each card in turn, and is asked to tell first what the card looks like or reminds him or her of, and second what details about the card make it look that way. Several formal systems have been devised for scoring subjects' responses to the Rorschach (Beck, 1952; Beck et al., 1961; Beck & Molish, 1967; Exner, 1974; Klophor & Davidson, 1962).

Previously, the Rorschach was frequently used by psychoanalysts to diagnose alcoholics, and studies were published that used the Rorschach to evaluate psychoanalytic precepts about the etiology of alcoholism (see Cox, 1985, pp.222-223). Generally, however, these studies do not provide strong support for psychoanalytic views about alcoholism.

Thematic Apperception Test. The TAT was published by Henry Murray and his associate Christiana Morgan in 1935 for the specific purpose of identifying people's unconscious psychological needs. The TAT consists of 29

cards depicting pictures or scenes (except for one blank, white card). People and objects depicted on the cards (e.g., a man, woman, house, tree, boat) are readily identifiable, but details such as exactly who the people are and what they are doing are ambiguous. A person taking the TAT is asked to make up as dramatic as possible a story about the scene on each card shown to him or her, telling what is happening in the scene at the present time, the events leading up to the present scene, and what the outcome will be. These details about the story must, of course, be projected from the person viewing the card. The TAT was used in seminal work by McClelland (McClelland et al., 1972) and Wilsnack (1974) to investigate men's and women's needs that are met by drinking alcohol.

Other tests of fantasy processes. Currently, projective tests are not widely used in research with alcoholics. However, other tests to assess relationships between drinkers' fantasy processes and their use of alcohol have been used recently. Cox and Klinger (1986), for example, used the Thought Sampling Questionnaire to study how frustration of goals and affective variables are related to impulses to drink alcohol. Segal, Huba, and Singer (1980) used the Imaginal Processes Inventory to study relationships between college students' imaginal processes and their initiation of drug use. Neither of these newer instruments in any sense is a projective test. We mention them here, however, because they are like projective tests in that they were designed to assess fantasy processes.

3.1.2 Objective Tests

The typical objective personality test consists of a list of items (e.g., statements) pertaining to personality, and the typical procedure is for a person taking the test to indicate whether or not each item applies to him or her. The scoring of these tests follows a routine procedure, so that any two persons who score them would arrive at exactly the same results. Thus, it is their scoring that causes them to deserve the designation "objective."

Unidimensional versus multidimensional tests. Unidimensional tests measure a single personality characteristic or a single dimension of personality. Common examples of unidimensional tests are adjective checklists and personality inventories (the latter consisting of self-statements) that measure such single dimensions of personality as depression, anxiety, and locus of control.

Multidimensional personality tests, on the other hand, provide a comprehensive description of personality by assessing its multiple dimensions. The personality test that has been used most frequently to study alcoholics is a multidimensional objective personality test, the Minnesota Multiphasic Personality Inventory (MMPI). The MMPI consists of 566 statements that are presented in a true/false format. These statements comprise 10 clinical scales (each of which measures a different dimension of personality) and three validity scales (which indicate how valid a test taker's responses are). Because the MMPI is used frequently with alcoholics (see Butcher & Tellegen, 1978), findings with it will be referred to frequently in this chapter.

Face validity versus empirical validity. The first objective personality tests (e.g., the Woodworth Personal Data Sheet; Woodworth, 1920) were

based on two assumptions. First, the items on these tests were assumed to measure the aspects of personality that were suggested by the wording of the items. Second, test takers were assumed to evaluate their own personality accurately and honestly. Because of these two assumptions, these tests are said to be face valid.

The assumptions underlying face-valid tests, however, present some difficulties. First, it is entirely possible that an item measures some aspect of personality other than the one that it appears on the surface to measure. For instance, a person's endorsement of the statement "I am always sad and blue" would appear at first to indicate that the person is depressed, but it could indicate instead that the person enjoys getting sympathy from other people. Second, the assumption that people evaluate their own personality accurately and honestly is questionable. For example, in certain situations people might be strongly motivated to conceal their weaknesses, but in other situations to present an unfavorable impression of themselves.

To overcome these difficulties, tests with empirical validity were developed, the best example of which is probably the MMPI. The psychologists who developed the MMPI (Hathaway & McKinley, 1943) first collected a large number of statements that psychiatric patients made about themselves. They then presented these statements as a "true/false" test to groups of psychiatric patients with clear diagnoses of various psychological disturbances and to "normal" people. Those statements that discriminated among the groups were retained for use on the MMPI and were assigned to one or more of the scales of the test. For instance, those items that schizophrenic patients answered differently from the other types of psychiatric patients and the "normal" respondents comprise the Schizophrenia Scale (Scale 8) of the MMPI.

The developers of the MMPI did not consider what the statements actually said in deciding whether or not to retain them on the test. Thus, even if a statement that discriminated schizophrenics from other people said nothing that appeared to be actually related to schizophrenia, it was still retained for use on the Schizophrenia Scale. In short, the items for the various scales of the MMPI were chosen empirically rather than on the basis of face validity. (As it turns out, some of the statements on the MMPI scales *do* and some *do not* reflect what one would rationally expect patients endorsing those statements to say about themselves.)

3.2 METHODOLOGY

In this section, we will summarize the various research methods for studying the personality antecedents of alcohol abuse and alcoholism, the personality characteristics of alcoholics and other heavy drinkers, and the effects of alcohol use and abuse on personality.

3.2.1 *Personality Antecedents of Alcohol Use and Alcoholism*

There are major practical difficulties in conducting a methodologically rigorous study of the personality antecedents of alcoholism. As a consequence, investigators have often relied on less than optimal procedures.

One such procedure has been simply to ask alcoholics to describe what

they were like before they became alcoholic (see Cox et al., 1983). The major disadvantage of retrospective accounts, of course, is that they are subject to memory distortions by the respondents. Nevertheless, the retrospective method has provided useful information, especially as it has generated hypotheses that could be more rigorously tested.

A second procedure that is less than optimal has been to analyse sources of information about alcoholics' personality that were recorded before their problem drinking began. This procedure, referred to as archival or archival longitudinal methodology (Cox et al., 1983), is more objective than retrospective methodology, but it has disadvantages of its own. For example, samples of alcoholics on whom archival data are available have been quite restricted, and it is unlikely that they are representative of alcoholics generally. The data themselves have also been restricted. They sometimes have not included results from standardized personality tests nor information about the topography of alcoholics' drinking behavior during their earlier life.

A superior, but not flawless, procedure has been to study the personality characteristics of persons who are at risk for developing problems with alcohol — a paradigm that has become increasingly popular in recent years. Two categories of high-risk subjects have been studied. They are (1) the biological sons of male alcoholics, who themselves are about four times as likely to become alcoholic as persons randomly chosen from the general population (Knop et al., 1984), and (2) young male drinkers whose personality characteristics resemble those of adult male alcoholics (e.g., Sher & Levenson, 1982). In spite of the increased likelihood of high-risk subjects' becoming alcoholic, there can, of course, be no guarantee that persons with a similar pattern of personality characteristics actually will become alcoholic. In fact, one would expect a bimodal distribution among subjects "likely" to become alcoholic, viz., those who do and those who do not subsequently become alcoholic.

From a methodological standpoint, studies that follow the prospective longitudinal design are the most desirable. Prospective longitudinal studies of alcoholism follow subjects from a point early in their lives to a much later time, after some of them have become alcoholic. These studies are able to identify the personality precursors of alcoholism more definitively than those following retrospective, archival, or high-risk methodology. In spite of the soundness of their methodology, however, prospective longitudinal studies are enormously time-consuming and expensive to conduct. Thus, we can appreciate the decision by some investigators (e.g., Jessor & Jessor, 1977) to follow their subjects for relatively brief periods of time, thereby identifying the personality precursors of initial problem drinking rather than alcoholism.

3.2.2 Personality Characteristics of Alcoholics and Other Problem Drinkers

The personality characteristics of alcoholics have sometimes been explored through the clinical case-study method. Clinicians using this method have published their clinical impressions about the personality dynamics in alcoholism that they gained from the alcoholic clients whom they saw in individual psychotherapy. In addition to clinical impressions, these reports

have sometimes included information obtained from personality tests, particularly projective tests such as the Rorschach Inkblot Test.

The typical procedure for identifying the personality characteristics of alcoholics, however, has been to administer personality tests to an entire group of alcoholics undergoing treatment for alcoholism and to report the results in the form of group averages. The subjects of these studies have often been male United States citizens hospitalized in public institutions. Since these subjects probably include only a small proportion of persons with severe alcohol problems, there is a danger in generalizing beyond these samples. Moreover, the practice of averaging the personality test scores of all patients in a treatment program as if they represented a homogeneous group has been called into question (Jackson, 1983).

A third research procedure has been to study the personality characteristics of heavy users of alcohol, including problem drinkers not diagnosed as alcoholic. Research with these subjects has provided valuable insights into the personality dynamics that underlie heavy consumption of alcohol. Again, however, caution must be exercised in generalizing beyond samples of heavy users, whose motivation for alcohol use may be different from that of diagnosed alcoholics.

3.2.3 Effects of Alcohol Use and Abuse on Personality

There are two ways to study how alcohol alters the personality of persons who imbibe it. One way is to note changes that occur from the sober to the intoxicated state. The other way is to assess how people's habitual consumption of alcohol affects their personality during the time that they are sober. In the latter case, the effects of drinking are cumulative and need to be monitored during a long period of time.

Considerable research has been devoted to clarifying the changes in personality that accompany intoxication. Two types of effects have been assessed: the "acute" effects of alcohol (the changes that occur during a single episode of intoxication) and the "chronic" effects of alcohol (the changes that occur during extended drinking sessions that last as long as several weeks). Identifying the acute and chronic effects of alcohol on personality has been considered important in elucidating people's motivation for using and abusing alcohol. Because studies of intoxicated behavior have often been designed to test the tension-reduction hypothesis of alcohol consumption, and since "tension" reflects negative affect, subjects' affective responses to alcohol have been the primary dependent variables in these studies.

The acute and chronic effects of alcohol have been investigated by administering alcohol to both alcoholics and social drinkers in experimental drinking situations. A variety of strategies have been used for administering the alcohol, and a variety of independent variables have been manipulated. Test sites have ranged from "sterile" laboratory to "naturalistic" ward environments for hospitalized alcoholics, to simulated parties for social drinkers. A significant advancement has been the use of the balanced placebo design (Marlatt & Rohsenow, 1980), which employs four groups of subjects, crossing the beverage (alcoholic or nonalcoholic) that subjects are led to believe they

are consuming with the beverage (alcoholic or nonalcoholic) actually consumed. This design allows the pharmacological effects of alcohol and the psychological effects due to subjects' expectancy to be separated.

It is far easier to identify the acute and chronic effects of alcohol on personality than the consequences that extended alcohol use has during the sober state. Nevertheless, several procedures have been used for the latter purpose. One procedure is to observe changes that occur when people who regularly consume alcohol abstain for a period of time. This situation occurs when alcoholics detoxify and enter treatment, but the procedure has been undertaken experimentally with social drinkers who voluntarily agree to abstain. In the case of detoxified alcoholics, the abrupt (negative) changes that occur with the removal of alcohol signify acquired dependence on alcohol, but the subsequent gradual (positive) changes seem to reflect the reversible, cumulative effects of alcohol. However, it is difficult to know the extent to which observed changes in personality are due directly to the removal of alcohol and the extent to which they are due indirectly to changes in the person's life situation that accompany abstinence. Finally, when it is possible to do so, comparing the personality of alcoholics before and after they became alcoholic provides clues about the effects of alcohol and an alcoholic lifestyle on personality.

4. BOUNDARIES OF THE THEORY

It has been clearly demonstrated that personality plays a major role in the development and maintenance of alcohol problems (see, for example, Jessor & Jessor, 1977, p.133). In §5, we will discuss the specific ways in which personality factors predate and coexist with alcohol problems, as well as the effects that alcohol has on personality.

Despite its important role, however, personality is only one of several categories of interacting variables that must be addressed in accounting for how alcohol problems develop and are maintained. Other factors include: (1) biological variables, such as a person's biochemical reactivity to alcohol; (2) psychological variables other than personality, such as a person's expectancy about the effects that drinking alcohol will have on him or her; (3) environmental variables, such as the availability of alcohol and the degree to which drinking is promoted in a particular environment; and (4) sociocultural variables, such as the drinking practices instilled by the culture in which a person lives. In §7, we will elaborate on how these variables interact with personality variables to account for the excessive use of alcohol. We will see that the weight that can be attributed to the different personality and nonpersonality factors varies greatly, both among individual alcoholics and within particular alcoholics from one point in time to another. Hence, the boundaries of a personality theory of alcoholism are not absolute.

Psychologists who advance a personality theory of alcoholism must acknowledge the contribution to alcoholism of these other categories of

variables. However, psychologists leave the task of accounting for the specific ways in which the other variables contribute to alcoholism to scientists from other disciplines. A full understanding of the complexity of alcohol problems will be realized, therefore, only through the joint efforts of scientists from multiple disciplines.

5. RESEARCH RESULTS AND NEEDS

The prodigious literature on the personality correlates of alcoholism has recently been reviewed by Barnes (1983), Cox (1983, 1985, 1987), and Lang (1983). Thus, the present section will summarize the major themes that are apparent from the research findings, rather than provide an exhaustive review of individual research studies.

5.1 PERSONALITY ANTECEDENTS OF ALCOHOL USE AND ALCOHOLISM

When male alcoholics have been asked to describe what they were like before they became alcoholic, they typically have reported that they were hyperactive, impulsive, and antisocial (see Cox et al., 1983). Although retrospective accounts must be accepted cautiously, alcoholics' self-descriptions have been found to coincide with the results of studies using more objective methodology.

One of the studies substantiating the retrospective reports was conducted in Minnesota and utilized archival data from the MMPI. Since most studies of alcoholics have also utilized the MMPI, the Minnesota archival study is valuable for directly comparing "prealcoholics" and alcoholics. The investigators studied data from: (1) prealcoholics and matched control subjects, the majority of whom were assumed not to have become alcoholic (Hoffmann et al., 1974; Loper et al., 1973); and (2) the MMPI protocols of male prealcoholics and protocols repeated an average of 13 years later when they entered treatment for alcoholism (Kammeier et al., 1973). The control subjects consisted of randomly selected classmates of the prealcoholics.

Comparing the MMPI profiles of the prealcoholics and their classmates, the investigators found that the configuration of the two profiles was very similar, and neither of them indicated disturbance or maladjustment. Nevertheless, the prealcoholics scored significantly higher on three of the standard MMPI scales (Psychopathic Deviate, Mania, and the F validity scale) than their classmates, as well as higher than their classmates on various MMPI derived scales designed specifically to detect alcoholism. The authors interpreted these differences as indicating that the prealcoholics were more impulsive, nonconforming, and gregarious than their classmates, although they were not more maladjusted or psychologically distressed. On the other hand, by the time that these same individuals had entered treatment, they were elevated on the MMPI scales that measure depression and anxiety: the Depression scale and the Psychasthenia scale. Various other archival studies (see Cox et al., 1983) provide substantial support for the findings of the

Minnesota study, usually describing prealcoholics as nonconforming, aggressive, and hyperactive.

Confirmation is also provided by the prospective longitudinal studies that have examined the personality characteristics of adolescents who later became problem drinkers. These studies have consistently found that future problem drinkers are independent and rebellious, and that they do not subscribe to conventional societal values (Jessor & Jessor, 1977, 1978; Wingard et al., 1980; Zucker, 1979; Zucker & Noll, 1982). Similarly, high-risk studies have described their subjects as impulsive and aggressive (Knop et al., 1984; Knop et al., 1985). On the other hand, future alcohol use and abuse have often *not* been predicted by low self-esteem, negative affect, or other indicants of psychopathology (Knop et al., 1984; Knop et al., 1985; McLaughlin et al., 1985; Sutker, 1982).

In summary, retrospective, archival, prospective, and high-risk studies all indicate that some groups of persons who in the future will become problem drinkers or alcoholics have personality characteristics that distinguish them from other groups of people. Prospective longitudinal studies that follow their subjects over extended periods of time are now needed in order to develop typologies of prealcoholic personality characteristics based on patterns of drinking behavior.

5.2 PERSONALITY CHARACTERISTICS OF ALCOHOLICS AND OTHER PROBLEM DRINKERS

The personality characteristics that have been found to distinguish alcoholics from other persons can be grouped into several broad categories that we will describe in this section. Additionally, we will delineate alcoholic personality subtypes, a topic that has recently been a focus of interest.

5.2.1 Nonconformity, Impulsivity, Reward Seeking

The personality characteristics that distinguished alcoholics from other people before their problems with alcohol began are also apparent after the onset of their problem drinking. We reach this conclusion on the basis of results from two personality tests that have been administered to alcoholics and other problem drinkers: the MMPI and the Sensation Seeking Scale (SSS).

Scale 4 of the MMPI. On the MMPI, alcoholics typically achieve their highest score on scale 4 (Psychopathic Deviate). That alcoholics are elevated on scale 4 is, in fact, a firmly established finding (see Cox, 1985, p.225). Scale 4 was developed to measure the personality characteristics of persons who show

disregard for social customs and mores, an inability to profit from punishing experiences as shown in repeated difficulties of the same kind, and an emotional shallowness in relation to others, particularly in sexual and affectional display The content [of the items on scale 4] ranges widely, reflecting the alienation of the person from his family and the extension of difficulties to school and to authorities generally. Some of the items involve frank admission of personal limitations, poor morale, and sexual troubles. At the same time, . . . there are also items involving denial of social shyness and assertion of social poise and confidence. (Dahlstrom & Welsh, 1960, pp.60-62).

Whereas persons who are highly elevated on scale 4 might show the nonconformity and independence described by Dahlstrom and Welsh, persons who are moderately elevated might simply approach social norms with a critical attitude. In any event, alcoholics' elevation on scale 4 is generally interpreted as indicating that they are alienated from the conventional mores of society and that they act impulsively to obtain immediate gratification of their impulses. Their inability to profit from experience seems to have gotten them into difficulties in various areas of their life. Despite the fact that they appear gregarious and socially adroit, they have difficulty forming satisfying, committed relationships with other people.

MAC Alcoholism Scale. There have been many attempts to derive scales from the MMPI that would serve the specific purpose of distinguishing alcoholics from nonalcoholics. One of these scales is the MacAndrew Alcoholism (MAC) scale, which consists of 49 MMPI items (excluding two items that pertain to alcohol use). The MAC scale was developed by MacAndrew (1965) to distinguish male alcoholics from male nonalcoholic psychiatric patients, and the fact that it does so successfully indicates that the scale taps male alcoholics' distinctive personality characteristics rather than general psychopathology. The MAC scale has been cross-validated with diverse samples of alcoholics, and has become the most thoroughly researched of the many alcoholism scales that have been constructed (see Cox, 1985).

The MAC scale, moreover, is the most successful scale for distinguishing alcoholics from nonalcoholics, and it has become widely used diagnostically in alcoholism treatment centres. With its increasing use by the psychometrically untrained, however, there is a danger that the MAC scale is being misused. It is not intended to be a definitive indicator of whether an individual is or is not alcoholic. The scale "misclassifies" approximately 15% of alcoholics as false negatives and approximately 15% of nonalcoholics as false positives (see MacAndrew, 1983).

From this "misclassification," MacAndrew developed a typology of alcoholics. He designated alcoholics who score high on the MAC scale as "primary alcoholics" and described them as "reward seekers" who are marked by boldness, aggression, and hedonistic tendencies. By contrast, MacAndrew designated the approximately 15% of alcoholics who do not score high on the MAC scale as "secondary alcoholics" and described them as "punishment avoiders" who are characterized by fear, reticence, and constricted interests. Although both primary and secondary alcoholics are depressed and anxious, MacAndrew hypothesizes that "it is their reaction to tension and depression that distinguishes them. Primary alcoholics drink and respond both actively and impulsively (drunken acting out), while secondary alcoholics appear to drink in the service of palliation" (MacAndrew, 1983, p.81).

Sensation Seeking Scale. The evidence from the MMPI regarding alcoholics' orientation to the world is amplified by the Sensation Seeking Scale. The current version of the scale (SSS V; Zuckerman, 1979) consists of 40 forced-choice items that provide scores on Total Sensation Seeking, Thrill and Adventure Seeking, Experience Seeking, Disinhibition, and Boredom Susceptibility. The scale, of course, has not been used as extensively with alcoholics

and other problem drinkers as has the MMPI, but a conclusion drawn from the existing research is that young, heavy alcohol users tend to score high on the Disinhibition Subscale, whereas older alcoholics tend to score high on the Boredom Susceptibility Subscale (Zuckerman, 1979).

5.2.2 Negative Affect and Low Self-Esteem

Various psychometric instruments have been used to evaluate the self-esteem of alcoholics. These include self-concept scales from multidimensional personality inventories such as the Barron Ego Strength scale from the MMPI, adjective checklists, Q-sorts, self-concept scales and inventories, and quite frequently the Tennessee Self-Concept Scale. The use of these instruments has consistently indicated that alcoholics (particularly female alcoholics) have low self-esteem (see Cox, 1985). Alcoholics' low self-esteem often manifests itself as a wide discrepancy between the way in which they view themselves (the self) and the way that they would like to be (the ideal self). Thus, a major goal in psychotherapy with alcoholics is to enhance their feelings about themselves (Curry & Marlatt, 1987).

Alcoholics' affect has been evaluated primarily with rating scales and various self-report measures, including the affective scales from the MMPI and tests designed specifically to measure affect and emotionality. The latter include various adjective checklists and mood scales and inventories (see Freed, 1978) and the currently popular Profile of Mood States (McNair et al., 1971). Assessments of alcoholics with these instruments has shown them to be emotionally reactive and to have strong negative affect (see Cox, 1987). For example, on the Neuroticism scales of the Eysenck Personality Inventory and Eysenck Personality Questionnaire (which measure general emotionality, worry, and tension), alcoholics have consistently scored higher than nonalcoholics. On the MMPI, alcoholics have typically been elevated on the Depression and Psychastenia scales (which measure depression, anxiety, and worry), and the degree of these elevations is exceeded only by their elevation on the Psychopathic Deviate scale. The severity of these symptoms, moreover, varies directly with the degree of alcohol dependence (Skinner & Allen, 1982).

Considering the consistency and intensity with which alcoholics experience negative affect, it is not difficult to see why they are sometimes diagnosed as having affective depressive disorders (Mayfield, 1985). Among problem drinkers, on the other hand, the relationship between drinking and depression is far weaker than among diagnosed alcoholics (Midanik, 1983). This difference suggests that depression may be a consequence rather than a precursor of problem drinking. We will return to this issue in a later section.

5.2.3 Cognitive/Perceptual Style

Alcoholics' cognitive/perceptual style has been studied along three dimensions: psychological differentiation, locus of control, and stimulus intensity modulation. In all three cases, differences have been found between the cognitive/perceptual style of alcoholics and nonalcoholics.

Psychological differentiation. Psychological differentiation refers to the degree to which people characteristically separate the self from the nonself (the field) in their cognitive/perceptual style. The separation has been concep-

tualized as unidimensional, with *field-independent* people relying on stimulus cues within themselves as their primary referent, and *field-dependent* people relying on stimulus cues in the external world (the field). Witkin and his associates (Witkin et al., 1954, 1962) devised various laboratory and paper-and-pencil tests to measure personality and perceptual relationships, including the Body-Adjustment Test, the Rod-and-Frame Test, and the Embedded Figures Test.

In the Body-Adjustment Test, persons seated within a small, tilted room are required to adjust their seat until they perceive their body to be in an upright position. Field-independent people are able to adjust their bodies to the true upright position, whereas field-dependent people adjust their bodies according to the axes of the surrounding room. In the Rod-and-Frame Test, subjects seated in a dark room are required to bring an illuminated tilted rod (that is surrounded by a tilted illuminated frame) to its upright position. Field-independent persons are able to perform the task accurately, whereas field dependent persons tend to align the rod with the frame. In the Embedded Figures Test, field-independent people are able to point out simple figures embedded in complex designs, whereas field-dependent people tend not to be able to do so.

The implications of psychological differentiation for interpersonal behavior have been the focus of subsequent research (Witkin & Goodenough, 1977). The general finding has been that field-dependent persons are more interpersonally oriented than are field-independent persons, although field-dependent persons do not show a pervasive psychological dependence on other people, as the Witkin group originally thought.

The Witkin group discovered that alcoholics are field-dependent: on the tests named above, alcoholics are unable to perform as well as nonalcoholics. Subsequent studies have consistently replicated this finding (Goldstein, 1976; Sugerman & Schneider, 1976). However, its implication for the etiology of alcoholism is not clearly understood at this time. It is still equivocal whether field dependence precedes the onset of alcoholism and contributes to it or whether field dependence is a consequence of excessive use of alcohol (see Cox, 1985, p. 218).

Locus of control. Locus of control refers to people's perception of the source of control in their lives. The source may be internal (i.e., people may perceive themselves to be in control) or external (i.e., people may perceive agents external to themselves, such as luck or other persons or forces, to be the controller). Most studies of the locus of control of alcoholics and social drinkers have used Rotter's (1966) Internal-External (I-E) Locus of Control scale, although various other locus of control scales have been used as well (see Rohsenow, 1983a). The latter include scales that specifically measure the locus of control of drinking behavior.

Since alcoholics are unable to control their use of alcohol or other aspects of their lives, it seems intuitively apparent that they would have an external locus of control. In fact, studies using appropriately matched nonalcoholic comparison groups have consistently found alcoholics to be more externally controlled than nonalcoholics (see Rohsenow, 1983a). Moreover, among

samples of nonalcoholic drinkers, the more that people drink, the more external they tend to be in their locus of control (Barnes, 1983; Naditch, 1975; Rohsenow, 1983a).

Despite the fact that the average alcoholic is more externally controlled than the average nonalcoholic, alcoholics have been found to differ widely in their perceptions of control, according to various demographic variables and drinking practices (Cox & Baker, 1982a,b; Rose et al., 1978). Alcoholics who are more externally controlled have more problems associated with their drinking and respond less well to treatment than do alcoholics who are more internally controlled. However, an important unresolved question for future research is whether particular treatment modalities are differentially effective with alcoholics who are more internally controlled and those who are more externally controlled (see Rohsenow, 1983a).

Stimulus intensity modulation. Not only does alcoholics' style of perceiving the world differ from that of nonalcoholics, but the intensity with which they perceive stimuli differs as well. This conclusion was reached by Petrie (1967) in her work on stimulus intensity modulation.

Stimulus intensity modulation refers to the intensity with which people perceive painful or other stimuli impinging on them. Augmenters overestimate the intensity, reducers underestimate intensity, and moderates estimate intensity accurately. The stimulus intensity that a person experiences can be evaluated with the Kinesthetic Aftereffect Task, as well as with paper-and-pencil measures. On the Kinesthetic Aftereffect Task, blindfold subjects estimate the size of stimulus blocks that they have just felt.

Petrie (1967) discovered that alcoholics are augmenters, and her discovery has subsequently been confirmed by other investigators (see Barnes, 1983). Petrie also discovered that augmenters and reducers respond differently to alcohol: augmenters become less sensitive to kinesthetic stimuli after ingesting alcohol, whereas reducers are unaffected. Hence, Petrie hypothesized that alcoholics use alcohol in order to modulate the intensity of unpleasant stimuli. In fact, subsequent laboratory studies have shown that alcohol serves as an analgesic for alcoholics and that college students who derive a stimulus-modulating effect from alcohol habitually drink alcohol for its medicating effect (Brown & Cutter, 1977). This finding presents an intriguing avenue for further exploring alcoholics' and nonalcoholics' different reactions to alcohol.

5.2.4 *Alcoholic Personality Subtypes*

The personality characteristics described above have been commonly found among samples of alcoholics in treatment. However, it would be a mistake to assume that these characteristics apply equally to all alcoholics. They are found in different degrees and combinations among individual alcoholics. In fact, as we indicated in §2, the heterogeneity of alcoholics has long been recognized. Using multivariate statistical analysis, researchers have identified subtypes of alcoholic personalities, primarily with four personality inventories: the MMPI, the Differential Personality Inventory, the Personality Research Form, and the Sixteen Personality Factor Questionnaire (see Cox,

1979; Jackson, 1983; Morey & Blashfield, 1981; Nerviano & Gross, 1983; Skinner, 1982).

At least seven different alcoholic personality subtypes have been identified, but only two of these have been identified repeatedly (Morey & Blashfield, 1981; Skinner, 1982). These two subtypes show different patterns and functions of drinking and different problems associated with their drinking. Alcoholics composing one of these subtypes are described as *sociopathic*. They have drunk over longer periods of time, but more moderately and have encountered fewer problems associated with their drinking than have members of the second subtype. They seem to use alcohol as a means to obtain gratification impulsively. Alcoholics composing the other subtype are described as *distressed and neurotic*. They are heavier drinkers and have more severe impairments associated with their drinking than do members of the first subtype. They seem to use alcohol to cope with their distress. It should be noted that these two subtypes closely resemble the "primary" and "secondary" alcoholics identified by MacAndrew with the MAC alcoholism scale.

Finally, we should note that the research on gender differences in alcoholic personality characteristics and life experiences suggests that different subtypes of alcoholism might occur in different proportions among male and female alcoholics. Female alcoholics more often than male alcoholics report that psychological crises precipitated their drinking problems (see Benson & Wilsnack, 1983). Thus, the situation in which personality difficulties lead to the excessive use of alcohol appears to occur more frequently among women than men. The situation in which personality difficulties follow the excessive use of alcohol appears to occur more frequently among men than women.

5.3 EFFECTS OF ALCOHOL ON PERSONALITY

During the early 1960s, there was a radical departure from the established dogma that alcoholics should never be given alcohol to drink. At that time, several studies (Diethelm & Barr, 1962; Docter & Bernal, 1964; Mendelson et al., 1964) were conducted in which alcoholics were given alcohol to drink in experimental situations. This research was influenced by the tension-reduction hypothesis of alcohol consumption, and its goal was to assess objectively the effect of alcohol on personality. The initial research was followed by a series of studies during the 1960s and early 1970s from the laboratories of Mayfield (1968a, 1968b; Mayfield & Allen, 1967), Mello and Mendelson (see Mello, 1972), and Nathan (see Nathan & O'Brien, 1971). In each case, alcohol was administered to hospitalized alcoholics during prolonged drinking periods, typically lasting for several weeks.

The results of this research were quite surprising. Instead of showing that alcohol uniformly reduced tension, the results showed that tension, anxiety, and depression mounted as drinking continued. Subsequent research with both social drinkers and alcoholics has indicated that the acute effects of alcohol on affect actually can be quite variable; alcohol can either intensify or palliate negative affect or contribute to positive affect. The variable effects, however, vary systematically with such factors as the dosage of alcohol, the

setting in which drinking occurs, whether the blood-alcohol level is ascending or descending, and drinkers' prior experience with alcohol (Abrams, 1983).

In spite of the variety of actual effects, drinkers consistently expect that alcohol will affect them in positive ways (Beckman, 1980; Brown et al., 1980; Christiansen et al., 1982; Rohsenow, 1983b; Southwick et al., 1981). They apparently recall the immediate positive affective changes that occur when they drink and tend to be unaware of the delayed negative effects. Moreover, the positive affective reaction to drinking is experienced most strongly by persons whose personality places them at risk for developing alcohol problems (Sher & Levenson, 1982).

What cumulative effects does drinking alcohol have on people's affect when they are in the sober state? In order to answer this question, Birnbaum et al. (1983) asked their experimental group of female social drinkers to abstain from drinking alcohol for a period of 6 weeks. Their control group (consisting of subjects who habitually drank at levels equal to those of subjects in the experimental group) continued to drink in their usual manner during the same 6-week period. During the course of the 6 weeks, the experimental group showed significant decreases in depression, anger, and confusion, whereas the control group showed significant increases in the same measures of negative affect. Other studies (Aneshensel & Huba, 1983; Rohsenow, 1982b) have also found that chronic consumption of alcohol intensifies drinkers' dysphoria while they are in the sober state.

Studies measuring the affect of alcoholics in treatment lend further support to the conclusion that the cumulative effect of alcohol is to intensify drinkers' negative affect during the sober state. Thus, the intense negative affect that alcoholics typically experience when they enter treatment subsides considerably as they proceed through treatment and remain abstinent from alcohol (Overall et al., 1985). When the personality of alcoholics entering treatment is compared with their own personality prior to the development of their alcohol problems (Loper et al., 1973), we find negative affect only during the later point in time, again a suggestion that the observed negative affect results from the excessive consumption of alcohol. Nevertheless, it is difficult to know the extent to which the negative personality characteristics observed among alcoholics are directly caused by alcohol and the extent to which they are indirectly caused by the disruptive effects of excessive drinking on the nonchemical incentives that could be enjoyed in life.

The effect that alcohol has on alcoholics' affect appears to change during the course of their drinking experience. People who later in life will develop problems with alcohol often seem motivated to drink initially in order to enhance their positive affect. They are involved in impulsive, nonconforming, reward-seeking activities and do not appear to experience negative affect that they would need to control by drinking alcohol. However, as the cumulative negative effects of excessive drinking take their toll, problem drinkers begin to use alcohol in an attempt to counteract their negative affect. Problem drinkers also appear to be less able than other people to utilize nonchemical sources of positive affect as an alternative to drinking alcohol. Hence, by the time that alcoholics enter treatment, they exhibit considerable depression and anxiety.

6. PRACTICAL IMPLICATIONS

There are several potential practical implications of being able to identify the personality precursors of alcoholism, the personality characteristics of alcoholics, and the effects of alcohol on personality. For the most part, however, the full benefit of these practical implications has not been realized.

Being able to identify persons at risk for developing alcohol problems by their personality characteristics might help in the development of primary prevention strategies. It might prove beneficial, for example, to educate persons who are potential alcoholics to use alcohol properly, or to avoid it altogether. However, stigmatization of potential future alcoholics should be avoided, especially given the strong likelihood of identifying false positives through this approach.

Identifying personality characteristics of alcoholics might help to improve secondary and tertiary prevention techniques, enabling treatment personnel to know what to do best to help alcoholics overcome their difficulties. More significant strides, however, will probably come from attempts to match different subtypes of alcoholic personalities with their most effective treatment modality. Some attempts to accomplish this end have already been undertaken (Conley, 1981; Conley & Prioleau, 1983; Finney & Moos, 1979; Morey et al., 1984; O'Leary et al., 1980).

Finally, knowing the effects that alcohol has on personality, and which of these are sought by the drinkers, might enable us to educate people about how to obtain the positive effects that they desire through nonchemical means. Helping people to assess the effects of alcohol on themselves realistically might also be an effective strategy to treat and prevent alcohol problems. Drinkers usually greatly overestimate the positive consequences of drinking and greatly underestimate the negative consequences.

7. RELATION TO OTHER THEORIES

As we saw in §2, the original attempts to account for alcoholism emphasized a single etiology (e.g., a "disease," the "alcoholic personality," "tension"). By contrast, current approaches eschew univariate explanations and emphasize multiple influences on the development of alcoholism.

Cox and Klinger (in press a, b) have developed a personality/motivational analysis of alcohol consumption that takes into account the multiple variables that converge to determine people's decision to drink or not to drink at any particular moment in time. Although the model depicts the ways in which personality and non-personality variables interact to determine people's decisions about drinking, we leave to other theorists the task of deciding the precise manner in which the non-personality variables have their influence. The model is motivational in the sense that it assigns a pivotal role to people's expected affective consequences of drinking versus not drinking in determining their actual decisions about drinking.

According to the model, three categories of variables determine people's habitual use of alcohol: biochemical reactivity to alcohol, personality characteristics, and sociocultural/environmental factors. Each of these variables acts to promote drinking or not drinking. To the extent that people do use alcohol habitually, they develop conditioned appetitive reactions to alcohol, which in turn increase the likelihood of current affirmative decisions about drinking.

Two current variables influence decisions to drink or not to drink. The first includes situational factors related to the availability of alcohol and the degree to which the situation and the people in it promote drinking. The second current variable is the strength of people's current positive and negative affect, which are determined by the quantity and quality of their current positive and negative incentives and their expectations of acquiring or losing such incentives in the future.

The historical and current factors give rise to cognitive mediating events that lead to specific expectancies about the effects that drinking will have on affect. People hold expectations about how drinking will affect the way they feel both directly and indirectly by virtue of the impact of drinking on the other incentives in their lives. People also react affectively to their *expectations* about affective changes from drinking — reactions that further intensify the tendency to approach or avoid alcohol. The ultimate decision to drink or not to drink is made on the basis of whether the expected positive affective consequences of drinking outweigh those of not drinking.

Other theorists (e.g., Huba & Bentler, 1982; Jessor, 1983; Jessor & Jessor, 1977; Zucker & Noll, 1982) have also suggested specific ways in which the multiple causes of alcohol use and alcoholism interact with one another. Moreover, the interaction of the biological, psychological, environmental, and sociocultural determinants of alcoholism is the theme of several recent books in addition to the present one (Cox, *in press*; Galizio & Maisto, 1985; Lettieri et al., 1980; Levison et al., 1983).

8. CONCLUDING COMMENTS

Great strides have been made toward identifying and measuring the personality characteristics of alcoholics since the time forty years ago when the idea emerged that the "alcoholic personality" might be both necessary and sufficient for alcoholism to develop.

With regard to prealcoholic personality characteristics, much of the evidence is available through happenstance or through longitudinal studies covering limited time periods. Nevertheless, the converging evidence clearly indicates that there are distinctive personality characteristics that predate alcoholism. Samples of persons who later in life develop problems with alcohol have been characterized by impulsivity, independence, social adroitness, and rejection of conventional values. These people impulsively find gratification through alcohol, but they have difficulty working toward long-range goals that will bring enduring satisfaction.

The same personality characteristics that we saw among prealcoholics are also apparent among alcoholics in treatment. They are characterized by a low tolerance for frustration, unconventionality, and sensation seeking, and their relationships with other people are marked by social adroitness, but superficiality and lack of intimacy. In other respects, however, the personality characteristics of alcoholics are different from those of prealcoholics. Unlike prealcoholics, alcoholics have low self-esteem and show strong negative affect (depression and anxiety). Although the cognitive/perceptual style of alcoholics is characteristically different from that of nonalcoholics, it is unclear whether this distinctive style is a precursor or a consequence of alcoholism.

Future alcoholics appear to be strongly reinforced by the effects of alcohol. However, the cumulative direct and indirect effects of alcohol on the personality of the alcoholic are quite negative. Hence, we typically observe low self-esteem, depression, and anxiety by the time an alcoholic enters treatment.

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6. CLASSICAL CONDITIONING WITH ALCOHOL: ACQUIRED PREFERENCES AND AVERSIONS, TOLERANCE, AND URGES/CRAVING

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1. PRINCIPAL ELEMENTS OF THE THEORY

The administration of a psychoactive drug provides the opportunity for associations to develop between the effects of the drug and concomitant stimuli. Because alcohol is consumed as a beverage, taste and odor cues are salient candidates for such stimuli. However, less obvious stimuli also may enter into association with its effects—for example, the environmental context in which alcohol is consumed (e.g., a bar or party), or internal feelings such as loneliness, depression, or gaiety. Thus, it is possible that events registered by our sensory organs and feeling states are associated with alcohol's effects.

Recently the role of drug-associated stimuli has assumed an increasingly prominent position in explanations of many drug-related phenomena including tolerance, sensitization, withdrawal, acquired preferences/aversions, craving, and self-administration (e.g., Baker et al., 1987; Baker & Tiffany, 1985; Eikelboom & Stewart, 1982; Goudie & Demellweek, 1984; Ludwig & Wikler, 1974; Siegel, 1979; Sherman et al., 1984a; Stewart et al., 1984; Wikler, 1965). Moreover, it has been argued that *classical conditioning* constitutes a valid model to account for the effects of drug-associated stimuli in these alcohol-related phenomena. It is our intention to evaluate the relevance and

implications of classical conditioning with respect to three major phenomena associated with alcoholism: preferences and aversions for alcohol beverages, alcohol tolerance, and alcohol urges or craving. To a lesser extent we will also discuss the role of classical conditioning in alcohol withdrawal. These phenomena have important roles in the development and maintenance of alcoholism. For example: (1) The fact that gustatory preferences and aversions for alcohol beverages develop and change over the course of alcoholism suggests that changes in alcohol palatability guide not only the selection of beverage but also the concentration of alcohol (e.g., "neat" whisky over beer). (2) Increased consumption of alcohol has been tied to the development of tolerance to the drug—with tolerance more alcohol must be consumed to achieve the effects once obtained with smaller quantities. Clearly, factors influencing tolerance development or maintenance are likely to influence the amount consumed. (3) Craving or urges to drink are a principal psychological manifestation of alcoholism that is associated with increased likelihood of drinking. It is our hope that characterizing the role of classical conditioning in these phenomena will provide the reader with a useful perspective in understanding and treating alcoholism.

The adoption of a conditioning model implies that the factors affecting the acquisition, maintenance, and extinction of classically conditioned responses should similarly influence these drug-related phenomena. Additionally, the model implies that associations acquired by classical conditioning are different from associations acquired in other ways (e.g., through socially transmitted expectations). For instance, the behavioral effects of a classically conditioned association may differ from those of other associations in terms of type of resultant response, response topography, the relationship between the response and important environmental stimuli, the role of awareness, or, finally, merely in terms of their conditions of origin. Understanding these implications, and others concerning the processes mediating classical conditioning, requires an introduction to some of the concepts, basic phenomena, and issues in classical conditioning. We present a brief treatment of classical conditioning aimed at this objective. For more information we suggest treatments by Estes (1975), Hilgard and Bower (1975), Mackintosh (1974, 1983), Schwartz (1984), and, of course, Pavlov (1927).

2. HISTORICAL DEVELOPMENT AND INTRODUCTION TO CLASSICAL CONDITIONING

2.1 THE BASIC PARADIGM, TERMINOLOGY, AND BRIEF HISTORY OF DRUG CONDITIONING

The term "classical conditioning" is used to describe the kind of learning studied by the Russian physiologist Ivan P. Pavlov. Recall that in Pavlov's (1927) celebrated experiment the sound of a metronome was *repeatedly paired* with the presentation of a puff of food powder into the mouth of a hungry dog. Whereas presentation of the food powder elicited a salivary response the first time it was presented, the metronome did not. However, after numerous

pairings of the metronome and food powder, the metronome also elicited salivation. The stimulus that unconditionally elicited a response prior to exposure to the learning procedure, in this case food, was called the *unconditional stimulus* (US), whereas the stimulus requiring the “conditions” of the procedure before eliciting the response, in this case the metronome, was called the *conditional stimulus* (CS). Pavlov labelled the responses to these stimuli the *unconditioned response* (UR) and *conditioned response* (CR), respectively. He sometimes called the US the *reinforcer* in this conditioning procedure to emphasize its importance in producing behavioral change to the CS.

Although Pavlov primarily studied salivary conditioning in the dog, research in his laboratory demonstrated the applicability of the paradigm to conditioning with drugs. Pavlov noted that the simple administration of a drug could be conceptualized as a conditioning trial. He reports (1927, p.35) a study of Dr. Krylov in which dogs were repeatedly injected with morphine. The effects of morphine included nausea, secretion of saliva, vomiting, and sleep. After several pairings of the drug-administration procedure with morphine, the preliminaries of injection were sufficient to produce many of the symptoms of morphine — nausea, secretion of saliva, etc. The symptoms of morphine had become conditioned to the drug-administration procedures. Even the sight of the experimenter was sufficient to elicit these responses in some cases. In terms of the conditioning paradigm, the drug-administration ritual served as the CS, morphine the US, morphine’s effects were the URs, and finally, the acquired responses to the CS constituted the CRs. Thus, study of the relevance of classical conditioning to drug-related phenomena had roots in Pavlov’s laboratory. Other reports of classical, or Pavlovian, conditioning with drugs serving as USs date back to the 1920s and ’30s. Collins and Tatum (1925) and Kleitman and Crisler (1927) were among the first to report successful Pavlovian drug conditioning. These authors used morphine as the US and obtained salivary CRs isodirectional (in the same direction) to morphine’s effects. Subkov and Zilov (1937) appear to have produced the first evidence that drugs can also support the acquisition of a CR antagonistic to the drug’s effect using epinephrine as the US. These researchers found that dogs displayed a bradycardic (slowed heart rate) CR even though the UR was tachycardia (increased heart rate).

Abraham Wikler was probably the first to suggest that classical conditioning played a significant role in the development of motivation to use drugs. In 1948, he proposed a conditioning model of opiate addiction and relapse (Wikler, 1948). Wikler’s model anticipated many research findings yielded by recent psychopharmacological research. It comprised the notions that drug USs could support both drug isodirectional and drug antagonistic CRs, and that the conditioning of the latter constituted substrates of both tolerance and dependence. In addition to his theorizing, Wikler contributed some of the first compelling data indicating that withdrawal signs could be conditioned to environmental cues, and that the relief of withdrawal symptoms constituted a potent US, conferring a preference on associated stimuli (Wikler & Pescor, 1967; Wikler et al., 1971).

Wikler’s work has served as a basis for subsequent research on drug

preference learning, tolerance development, and conditioning models of urge acquisition. However, within each of these areas a small number of investigations provided a crucial impetus to further work on each topic. For example, John Garcia's research on taste preference learning, with the relief of apomorphine illness as the US, spurred research into the conditioning of taste preferences with either drug effects or withdrawal-relief as the US (Green & Garcia, 1971). Shepard Siegel's research (Siegel, 1979) constituted an important work on a Pavlovian model of drug tolerance, and Arnold Ludwig's research established an approach to assessing the potential contribution of Pavlovian associations to alcohol urges (Ludwig et al., 1974).

2.2 THE STIMULUS-REINFORCER DEPENDENCY

A cardinal feature of the classical conditioning procedure is that the presentations of the CS and reinforcer (US) do not depend on the organism's behavior. This contrasts with the operant conditioning paradigm in which receipt of the reinforcer depends upon response production. Frequently these differences are characterized in terms of the critical procedural dependency: in classical conditioning it is the stimulus-reinforcer dependency whereas in operant conditioning it is the response-reinforcer dependency. Despite these procedural differences, it has been persuasively argued that a single learning, or associative, process underlies the behavioral changes induced by these two conditioning paradigms (Bindra, 1969; Hull, 1943). However, we are swayed by the evidence marshalled by the two-factor theorists, who argue that the nature of the associative learning mediating behavioral changes in classical and operant conditioning are different (Konorski, 1967; Mackintosh, 1974; Rescorla & Solomon, 1967; Skinner, 1938). In classical conditioning we assume that the learned association generally is between the CS and some component of the US, whereas in operant conditioning it is between a particular response and the reinforcer, although with some kinds of classical conditioning procedures there is evidence that the CS becomes associated with a component of the UR instead of the US (cf. Rescorla, 1980).

2.3 STRENGTH OF ASSOCIATION

Factors that affect the strength of CS-US associations include: the timing between CS and US presentation, the number of conditioning trials, and the proportion of CS presentations that are followed by a reinforcer. With respect to CS-US timing, research shows that, in general, the sooner the US occurs after the CS, the greater the learning. However, some types of stimuli may produce learning despite lengthy CS-US intervals. For instance, animals may associate gustatory CSs with illness-inducing treatments even if such treatments are delayed up to 24 hours after the CS (Smith & Roll, 1967). In addition, if the US is presented prior to the CS ("backwards conditioning"), the CS may actually inhibit the display of the CR rather than elicit it (Rescorla, 1969). With respect to the number of conditioning trials, it is generally true that number of conditioning trials and CR strength (magnitude and/or probability) are positively related; however, performance tends to be asymptotic (cf. Mackintosh, 1974). Finally, *partial reinforcement* (presenting the US on only a portion of the CS trials) tends to retard the rate of CR acquisition (cf. Mackintosh, 1974).

Once conditioning has been established, the CR may be reduced by presentation of the CS alone. This procedure is referred to as *extinction*. The greater the number of CS-alone trials, the greater the extinction effect. Interestingly, with time following extinction there is some recovery of the CR, referred to as *spontaneous recovery* (Prokasy, 1958). This phenomenon is robust, but the CR may be attenuated further with subsequent extinction trials.

2.4 CONTINGENCY LEARNING

A primary concept about learning that has lasted from the British Associationists until quite recently, is that learning is produced by the simple co-occurrence, or contiguity, of two events. Indeed, this notion was endorsed by Pavlov, who argued that conditioning was due solely to the repeated co-occurrence of the CS and US. However, Rescorla (1967) demonstrated that simple temporal contiguity was not sufficient to account for Pavlovian learning. Rescorla suggested that classical conditioning was best accounted for by the *contingency* between CS and US events: that is, the probability that the CS and US will co-occur, relative to their independent occurrence.

A contingency account of classical conditioning helps to explain the detrimental effects of CS or US pre-exposures on subsequent conditioning. If animals are exposed repeatedly to the CS prior to pairing of the CS and US, acquisition of a CR will be delayed (Lubow, 1973). This phenomenon, known as *latent inhibition*, can be attributed to the fact that the CS pre-exposure results in a poor CS-US contingency (cf. Mackintosh, 1983). This same explanation can account for the attenuation of learning that results from US pre-exposures (Cannon et al., 1975). Perhaps the simplest way to conceive of the CS-US relationship is that Pavlovian learning will occur as a function of the extent to which the CS, and only the CS, is an accurate signal of the US.

2.5 RANGE OF STIMULI

Stimuli perceived through almost any sort of sensory modality can function as CSs. Effective CSs may be olfactory, orosensory, tactile, auditory, thermic, or visual in nature. In addition, there is considerable evidence that a variety of internal, or interoceptive, stimuli can serve as effective CSs. Bykov's classic illustration of an interoceptive CS was the application of glucose or hydrochloric acid to intestinal loops (Bykov, 1957). A comparable heterogeneity holds with respect to the range of stimuli that may serve as effective USs: exteroceptive stimuli such as shock, noise blasts, or temperature changes are effective, as are interoceptive stimuli such as emetic drugs or vitamin repletion (e.g., Seligman & Hager, 1972).

Interestingly, evidence supports the notion that interoceptive and exteroceptive CSs are not equally associable with all classes of US. For example, Garcia (Garcia & Koelling, 1966; Garcia et al., 1970) and his colleagues have shown that for the rat, interoceptive taste cues are more likely to be associated with USs producing internal malaise (e.g., an emetic) than USs producing peripheral pain (e.g., electric shock to the paws). In contrast, this relationship is reversed when the CS is an exteroceptive cue; that is, an exteroceptive "bright, noisy" CS is more readily associated with peripheral pain than internal malaise.

Garcia has argued that these associative biases reflect the effects of selective pressure on the rat's adaptation to its ecological niche where tastes are more likely to be better predictors of internal malaise than are exteroceptive stimuli. The converse is true with respect to painful peripheral insult. Evidence suggests that a similar relationship between interoceptive and exteroceptive CSs and internal malaise is obtained with humans (cf. Baker & Cannon, 1982; Sherman et al., 1984a).

2.6 RETENTION OF THE CR

In the absence of extinction trials (CS-alone trials), Pavlovian CRs may be retained for extraordinary time periods. Kimble (1968) cites the 2-year retention of a conditioned motor response by sheep, and the 30-month retention of a leg-flexion response by a dog. Such retention information is instructive if one is interested in accounting for persistent effects of drug use such as the appearance of cigarette urges after years of abstinence or the 9-month retention of tolerance (Cochin & Kornetsky, 1964).

2.7 LEVELS OF PROCESSING

Two extreme views of Pavlovian learning are that: (a) it is an automatic process that occurs similarly in the human and the mollusk, and requires little conscious processing or awareness; and (b) Pavlovian learning, at least in humans, does not occur without conscious or controlled processing and awareness, and is greatly affected by information transmitted through nonconditioning routes. Posner and Snyder (1975) propose that automatic processing differs from controlled processing in that only in the former does processing: (a) occur without intention; (b) occur without awareness; and (c) interfere little with other ongoing mental activity, occupying little cognitive workspace.

Consider first the possibility that Pavlovian learning is automatic. There is evidence that Pavlovian learning in humans and invertebrates is similarly influenced by such procedures as extinction and partial reinforcement, and both humans and invertebrates display such phenomena as spontaneous recovery (Schwartz, 1984). If it is indeed the case that Pavlovian learning occurs similarly across highly varied species, it suggests that in humans such learning may not require much controlled processing. Indeed, Pavlovian learning can be shown to occur in animals when controlled processing is unlikely: for example, when animals are made unconscious through anesthesia (Rabin & Rabin, 1984; Roll & Smith, 1972; Weinberger et al., 1984). More direct evidence is provided in a recent review by Dawson and Schell (1985) concluding that human research has shown that the classical conditioning of eyeblinks or electrodermal responses occurs despite subjects' attempts to block, or resist, conditioning (e.g., Norris & Grant, 1948). Finally, Bernstein (1978) showed that humans acquired taste aversions despite their correct awareness that the taste CS did *not* cause the illness US. Thus, we see that in at least two important respects, Pavlovian learning in humans appears to involve automatic processing: learning occurs despite countervailing causal information, and when conditioning is consciously resisted.

In contrast to the above evidence, there are data suggesting that Pavlovian learning depends in some cases upon controlled information processing.

First, although Pavlovian CRs may be acquired despite subjects' attempts to block CR acquisition, such CRs are much weaker than those acquired with a cooperative subject (Dawson & Reardon, 1969). This suggests that associative or response processes involve controlled processing. Second, considerable research shows that if awareness is not a *sine qua non* of human Pavlovian conditioning, it certainly appears to be a prerequisite in many instances (e.g., Dawson & Schell, 1985; Maltzman, 1979). Third, numerous studies have shown that CR strength is greatly affected by information transmitted via a nonconditioning route (e.g., verbal instructions that extinction has begun; Wilson, 1968), suggesting that CRs are not automatically elicited, but rather are displayed as a function of a person's total appraisal of a response context. Finally, relevant to Posner and Synder's third characterization, Dawson et al. (1982) have recently shown that during the acquisition of a Pavlovian electrodermal CR, reaction time latency increases on a secondary nonconditioning task, a suggestion that the formation of Pavlovian associations requires the allocation of cognitive workspace, thereby interfering with other mental work.

What conclusions can we derive from the above basic conditioning research? Pavlovian conditioning does not appear to be dichotomous along the three dimensions presented. There is mounting evidence that human Pavlovian learning is a mix of processes, some of which seem "automatic" and others "controlled" (e.g., Dawson & Reardon, 1969; Dawson & Schell, 1985). Evidence of the heterogeneity of conditioning phenomena has led theorists to hypothesize that the nature of processes involved in Pavlovian learning will vary as a function of species (e.g., Razrin, 1965), learning task (e.g., Maltzman, 1979), nature of the US employed (Mandel & Bridger, 1973), or nature of CS (Garcia et al., 1974). For instance, Maltzman (1979) suggested that the type of learning typically encountered in the human learning laboratory might be characterized by voluntariness and a communicable awareness. However, Pavlovian learning of the sort that presumably produces phobias may be characterized by involuntariness, a lack of awareness, and countervailing attitudes and beliefs. The phobic reports fear of a snake *despite* the fact that he/she knows that the snake is innocuous and the US (e.g., a bite) may never occur. Consistent with this are findings by Hugdahl and Öhman (1977) showing that electrodermal CRs acquired to presumed phobic stimuli are refractory to instructions that extinction is in effect.

It may be that most learning phenomena are supported by diverse underlying processes, some conforming to "automatic" criteria and others to "controlled" criteria. This diversity may reflect the fact that Pavlovian learning in humans, like other complex human nervous system functions, occurs via hierarchically organized neural systems. The operating principles of some of these may conform to automatic processing characteristics while those of others do not. Pavlovian learning cannot be restricted on an *a priori* basis to a particular level of awareness, level of volition, or even level of consciousness. Yet, all of these variables undoubtedly affect the rate and durability of Pavlovian learning. Thus, although we may define Pavlovian learning concisely on the basis of procedures, it is much more difficult to identify the boundaries of the cognitive or information processing procedures that influence the

development of Pavlovian associations, or the production of the CR. One implication of the latter perspective is that even if an addiction phenomenon (e.g., urges) has Pavlovian origins (CS-US pairings), a variety of procedures other than extinction might influence the magnitude or persistence of conditioned responding, such as moral suasion, information that further drug use will be fatal, or increased skill in coping with urges.

2.8 WHAT IS LEARNED?

The question of what is learned in Pavlovian learning is complex and has not yet been resolved. However, we believe that it is important here to ensure that the reader is aware that boundary conditions on the CR, assumed to exist because of early notions regarding the nature of Pavlovian learning, are no longer widely endorsed. Early hypotheses about the nature of Pavlovian learning, including Pavlov's, emphasized that the organism learned to respond to the CS as if it were the US. An implication of such stimulus "substitution" theories is that the CR must be highly similar to, if not identical to, the UR. However, considerable research now indicates that stimulus substitution theories are certainly incomplete, if not incorrect. Cognitive theories of Pavlovian learning emphasize that organisms can acquire an expectation of the US and that the organism apparently may engage in preparatory responses consistent with this expectation (Tolman, 1932). This view is compatible with findings that in many cases the CR and the UR are dissimilar—for example, deceleratory cardiac CRs where the UR is acceleratory (e.g., Obrist, 1968; Somsen et al., 1983). Similarly the UR to electric footshock to the rat is rapid scurrying about, whereas the CR is freezing (Fanselow, 1982).

2.9 FUNCTIONAL SIGNIFICANCE OF CLASSICAL CONDITIONING

Pavlov (1927) viewed classical conditioning as a laboratory model of behavioral adaptation to the US. This point is pursued because it may have heuristic value in conceptualizations of the role of classical conditioning in pharmacologic effects. Konorski (1967) distinguished between two types of conditioning that influence the form of the CR: one form resulted in the *preparatory* CR, a diffuse expression of a general emotional state sometimes evidenced as approach or avoidance of the CS, and the other resulted in the *consummatory* CR, a precise, discrete reflex specific to the nature of the US. Mackintosh (1974) has argued that the preparatory CR reflects the association between the CS and the motivational attributes of the US, whereas the consummatory CR reflects the associations with the specific sensory attributes of the US. Konorski assumed that both preparatory and consummatory CRs occurred in the same conditioning experiment and that the preparatory, motivational CR was a necessary background for development of the specific consummatory CR. These two forms of the CR would be expected to have different, but complementary, adaptive significance. The preparatory CR would be expected to exert a motivational effect that would influence the organism's approach/avoidance disposition, whereas the consummatory CR would be expected to prepare the organism, behaviorally or physiologically, for the specific sensory features of the US. For a hungry dog, a CS associated with a food US might be expected to elicit both approach responses and a specific

salivary response. An organism that has learned this Pavlovian association would be expected to have the advantage of (a) approaching the potential source of food and (b) being prepared for immediate ingestion.

Thus, theoretical analyses of the adaptive significance of preparatory and consummatory CRs suggest that with addictive drug USs the likely effects of classical conditioning would be (a) an altered approach/avoidance gradient with respect to drug-associated stimuli (e.g., Crawford & Baker, 1982; Sherman et al., 1980a,b), and (b) an enhanced ability to cope with the drug stimulus (Siegel et al., 1982).

3. CHARACTERISTIC RESEARCH METHODS

3.1 THREE CRITERIA FOR ASSESSING A CLASSICAL CONDITIONING MODEL

With this basic introduction to classical conditioning completed, the criteria typically used to assess the applicability of a classical conditioning model to alcohol-related phenomena may now be introduced in a more meaningful context. We have conceptualized three major criteria:

1. Are the essential procedural elements of classical conditioning present in the case of drug or alcohol self-administration? Specifically, are stimuli present that may serve as CSs and USs? Does their relationship conform to a classical conditioning contingency? Is there a CR? Does its elicitation bear on the particular phenomenon?
2. Do changes observed across repeated drug exposures, or across repeated exposures to drug-associated cues, conform to predictions generated by a classical conditioning model? In particular, do observed changes conform to the parametric features of Pavlovian learning as characterized in basic research?
3. Finally, is there any evidence that the same processes that subserve classical conditioning also produce changes in response to drug-associated stimuli? That is, can the nature of the processing, what is learned, and the adaptive significance be conceptualized along dimensions within the Pavlovian paradigm?

We suggest that, taken together, these three criteria form the basis for assessing the applicability of a Pavlovian framework for a particular drug-related phenomenon — they serve as “inclusion” criteria. They may also be useful for excluding alternative hypotheses. For example, consider the following hypothetical model of alcohol tolerance. This proposed model suggests that tolerance is the result of physiological adaptation to alcohol, such as by enhanced enzymatic degradation of alcohol resulting from repeated alcohol metabolism. This physiological model asserts that tolerance is simply a consequence of repeated experience with alcohol. It does not predict that tolerance is specific to alcohol-associated stimuli, or that tolerance may be modulated by manipulations that would alter the strength of the association between such stimuli and alcohol, such as by partial reinforcement or extinction.

manipulations, as is indicated by criterion 2 above. Evidence that alcohol tolerance is influenced by such factors would call for the rejection of the proposed physiological model—at least as a complete account of tolerance. Thus, these criteria not only provide the opportunity for addressing the applicability of a Pavlovian framework but also suggest strategies for assessing the adequacy of alternative theoretical frameworks.

Demonstrating that a drug-related phenomenon meets all three criteria is a formidable task. Indeed, most investigators, as we shall see, have done well to validate empirically the first two criteria (e.g., Siegel, 1979). The third criterion has received less empirical attention because relevant theory has not clearly defined how these objectives should be tested within the context of classical conditioning with drugs. Very recently, however, some ideas have emerged for approaching the issue of mediating processes (e.g., Baker & Tiffany, 1985; Eikelboom & Stewart, 1982; Stewart et al., 1984). In any case, we will review these criteria with respect to preferences and aversions for alcoholic beverages, alcohol tolerance, and alcohol craving—all drug-related phenomena that have been cast in a classical conditioning framework.

3.2 GENERAL RESEARCH METHODS

In this section we describe the major associative control procedures common to nearly all studies of Pavlovian conditioning phenomena. This treatment surveys the major methodological concerns in the study of Pavlovian conditioning and provides a general background relevant to the specific methodologies for each of the content areas to be discussed here: that is, preferences/aversions, tolerance, and craving. Most of the research in the first two content areas has focused on the acquisition of conditioned effects and subsequent manipulations such as extinction. Ensuring that the effects of conditioning trials are due to associative factors has been critical in establishing a conditioning model of these phenomena. In contrast, the study of alcohol craving among alcoholics or heavy drinkers has been based on the assumption that drug-associated stimuli have already acquired their capacity to elicit craving via natural conditioning processes. However, in some research, implications of an associative framework are assessed, for example, by testing whether the craving CR is subject to extinction. Unfortunately, such tests only provide indirect evidence of a Pavlovian origin of craving.

Here we offer a limited treatment of the role of associative controls to illustrate the assumptions underlying the Pavlovian model. Lastly, we discuss problematic issues in identifying the specific process by which alcohol can act as a US.

3.2.1 *Control Procedures in the Study of Classical Conditioning*

The *sine qua non* of classical conditioning is that the emergent CR reflects a learned association between the CS and US. However, the conditioning procedure provides the opportunity for the development of responses to the CS, arising from non-associative processes, that may nonetheless be mistaken for the CR. That is, these responses are not attributable to the temporal pairing and/or contingency between the CS and US. Non-associative responses traditionally have been attributed to the effects of sensitization or pseudoconditioning (e.g., Gormezano & Kehoe, 1975).

Sensitization refers to the possibility that responses elicited by the CS are sensitized, or potentiated, by exposure to the US. For example, the sudden presentation of a tone may elicit a startle reaction of which one component is the eyeblink. Typically, the magnitude and reliability of the eyeblink response to a tone is small. However, if a puff of air is administered to the eye (a US that potently and reliably elicits an eyeblink), sensitization would be evidenced by a stronger eyeblink to the tone. That this sensitization effect is non-associative is evidenced by the fact that the air puff need not be paired with the tone to produce the response.

The pseudoconditioned response refers to the possibility that the response elicited by the US may be elicited by other stimuli despite the absence of an association between them. In contrast to sensitization, the CS need not have a propensity for eliciting the target response. Pseudoconditioning may occur with *any* CS. For example, if a US was very potent or noxious, the presentation of any CS might elicit a startle response of which a component could be the target response. The “take-home message” with either sensitization or pseudoconditioning is the same: non-associative processes can contribute to the measurement of the CR and inflate the assessment of conditioning. With pharmacological USs the assessment of non-associative processes is critical to the accurate characterization of associative effects. Consider the following hypothetical case that borrows from the sensitization example above: If an organism is given alcohol, the drug may sensitize one or more response systems. For example, doses of alcohol sufficient to alter body temperature may make thermal reactivity to nonpharmacological stimuli more likely. If a component of the CS is stressful enough to elicit marginal changes in temperature (e.g., emotional hyperthermia, Briese & de Quijada, 1970), prior exposure to alcohol may sensitize this hyperthermic response. Thus, prior exposure to alcohol, *per se*, may be sufficient to potentiate responses to nonpharmacological stimuli (CSs) that naturally act on systems influenced by alcohol; such processes might lead us to incorrectly assume that the increased CR is exclusively due to associative factors.

Recent interest in the role of conditioning in alcohol tolerance underscores the necessity of assessing the contribution of non-associative factors. Although traditional views of tolerance have varied in terms of the precise mechanisms posited, they have been similar in suggesting that iterative exposures to drug, *per se*, were sufficient to produce tolerance. Clearly, if associative factors influence tolerance, the contribution of non-associative processes in tolerance development must be teased out with the use of an appropriate control procedure.

To assess the possible contribution of non-associative factors to the CR, various control treatments have been used. These have included: (a) a CS-alone control, (b) a US-alone control, (c) an explicitly unpaired control in which the CS and US are both presented but with interstimulus intervals long enough to ensure that the events are “unpaired,” (d) the backward control in which the onset of the US precedes onset of the CS, and (e) the random control in which the CS and US share no systematic temporal relationship.

The selection of a specific control procedure often reflects the

researcher's theoretical perspective on the necessary conditions for promoting conditioning. An investigator holding the theoretical view that the CS-US contingency, rather than contiguity, is necessary for learning is most likely to employ the random control. The random control, which may result in the chance contiguous pairing of the CS and US, is generally viewed as the most conservative control procedure, whereas the CS-alone or US-alone controls are generally viewed as the least conservative. In drug conditioning experiments it is rare to find use of the true random control procedure because: (a) typically so few trials are given that chance pairings could constitute a high proportion of the total CS and US events; and (b) the US and UR last such a long time. However, at least one study has employed an approximation of a random control in demonstrating conditioned morphine tolerance (Siegel et al., 1982).

The most frequently employed associative control in drug conditioning experiments has been based on the discriminative conditioning paradigm. In this paradigm, subjects are exposed to a CS+ (i.e., always followed by the US) and to a distinct CS- (i.e., never followed by the US). Successful conditioning is said to occur when the organism displays greater evidence of a CR in the context of the CS+ than the CS-. The assessment of non-associative effects is conducted by experimentally balancing the specific stimuli serving as the CS+ and CS-. A study by Siegel (1978) on the role of conditioning factors in tolerance to the hyperthermic effects of morphine illustrates such balancing and raises a general concern with the frequently used discriminative conditioning paradigm. In his study, one group of rats were administered morphine in a distinctive context (DC), a room with a white noise; a saline injection was given in the context of the homecage (HC) environment. For this group the DC served as the CS+ (DC/CS+) and the HC served as the CS-. For a second group, the DC served as the CS- (DC/CS-) and the HC served as the CS+. Thus, the discriminative stimuli were balanced with respect to the CS+ and CS-. The development of hyperthermic tolerance was measured by repeated assessments of rectal temperature in the DC environment for group DC/CS+. To equate for handling and possible stress, group DC/CS- were similarly tested but without drug. Following an equal number of CS+ and CS- trials, Siegel then administered morphine to both groups in the DC and assessed tolerance to the drug. Note that for one group this was the CS+, for the other it was the CS-. Consistent with Siegel's Pavlovian model of tolerance (discussed later in §4.2), group DC/CS+ displayed greater tolerance to the hyperthermic effects of morphine than did DC/CS-. It is to be noted that both groups had the same experience with drug and both environments — seemingly the only difference between the groups was the contingency between stimulus (CS+) and drug (US). Presumably, if the groups had been tested for tolerance in the HC, the relative degree of tolerance in the two groups would have been reversed.

However, we are tentative about drawing this conclusion because of recent evidence suggesting that non-associative effects related to stress and stress/drug interactions can bias the effects of drug conditioning experiments (e.g., Rochford & Stewart, 1987; Sherman et al., 1982, 1984b; Zelman et al., 1985). Note that in the above example, as well as in other studies following a

similar discriminative conditioning and testing protocol (e.g., in our own research, Sherman, 1979; Tiffany et al., 1983), only one discriminative cue (in this case DC) was paired with the potential stress of target response assessments (e.g., repeated rectal probes). Thus during acquisition training, one group may have received stress plus drug (CS+) in the DC (e.g., DC/CS+), whereas the other group received stress alone in the DC (e.g., DC/CS-). This is a potential problem for two reasons. First, it provides the opportunity for drug/stress interactions for group DC/CS+ but not DC/CS-. The effects of drug in the presence and absence of stress may differentially influence drug action (e.g., Zelman et al., 1985). Second, only group DC/CS- received stress-alone exposures in the CS-. Because stress effects may be conditioned, and it is known that conditioned stress effects may modulate drug effects (Sherman et al., 1984), the DC may have served to modulate the effect of morphine in a different manner for group DC/CS- than DC/CS+. Thus, although the two groups had the "same" treatments to the extent that both experienced the DC, HC, drug, and repeated target response assessments prior to testing, they differed with regard to the potential *interactive* effects of these stimuli.

This difficulty explains our tentativeness in drawing an associative interpretation of the above data. Fortunately for future research, the impact of these potential non-associative confounds can be assessed easily—simply testing rats for tolerance in both environments is sufficient. If symmetrical test results are obtained on the basis of CS+ (DC or HC), the results are not likely to be due to differential stress effects.

Of paramount importance in drug conditioning experiments is that both experimental and control groups receive equivalent exposure to the US prior to the assessment of conditioning effects; otherwise, non-associative change in pharmacological response could contribute to observed differences. Additionally, the frequency of exposure to the test environment should be controlled, because it is known that novel exposure to a test stimulus is stressful and this stress may yield effects that mimic the contribution of the CR to tolerance (Sherman, 1979). These considerations suggest CS-alone or US-alone controls are inappropriate for drug-conditioning research.

3.3 THE ALCOHOL US

Alcohol has a constellation of physiological and psychological effects (Jaffe, 1975). To name just a few of the more commonplace effects: Alcohol is a food with caloric density almost twice that of sugar; it is a recreational drug, apparently having affective and disinhibiting consequences that enhance the pleasure derived from social events; it is a sedative often used to "cope" with the disquieting effects of stress; it is a toxin, overdoses of which produce effects including visceral pain, dizziness, nausea, vomiting, and headache. These myriad appetitive and aversive effects may act individually or in concert as the US in any experimental paradigm, depending on dose, prior experience with the drug, and the circumstances of use. Researchers are encouraged to isolate which among the constellation of effects accounts for the observed consequences of drug administration. This issue is of clearest importance in the

study of the conditioned motivational properties of alcohol in which stimuli associated with it activate incentive systems supporting pursuit or avoidance of it. For example, consider the hypothetical case in which an experimental group of hungry rats is given a neutral taste CS mixed with a weak alcohol US. Compared to controls that receive unpaired presentations of the CS and alcohol, the experimental group displays greater consumption of the CS when it is presented alone. To which component of the alcohol US may we attribute this conditioned taste preference — the taste, odor, calories, or central effects of the drug?

Conditioning of nonmotivational properties of the US presents a similar problem. For example, if conditioned temperature changes are evidenced to an environmental CS paired with alcohol injected intraperitoneally (i.p.), it is unlikely that taste or odor account for this effect (although alcohol does have gustatory properties even when injected i.p.; Cunningham, 1978). It may be that a temperature regulation system has been conditioned, or the temperature change is secondary to a conditioned kinetic effect, or to a general conditioned emotional response — a response that might be conditioned with any motivationally significant US. Alternatively, while alcohol might directly influence a pyretic system, it could be doing so by changing central thermostats, or instead by bypassing central thermoregulators and affecting efferent mechanisms. Consequently, specification of the alcohol US remains a problem in any conditioning procedure. It should be noted that although this concern may be important to understanding the precise mechanism mediating alcohol conditioning, such specification is not necessary for using conditioning paradigms as a model for alcoholism. There are implications for treatment (e.g., extinction) even though we may not know which component or components of the US contribute to a conditioned effect. The research on conditioned alcohol effects has only begun to ask which component(s) of the alcohol US could account for a specific conditioned effect. Various strategies for isolating the locus of drug effects have been employed. These include varying the route of administration, varying dose, surgical manipulations, biochemical manipulations, and the use of control agents that duplicate one component of the alcohol US.

It is worth noting that the physiological distinction between the alcohol US and UR is sometimes difficult to make. Typically, with exteroceptive events, like electric footshock, the US refers to the afferent influence on the central nervous system (CNS), that is, the input to the CNS. The UR typically refers to the efferent consequences of CNS activation by the US, that is, output of the CNS in response to the US. However, with drugs, as Eikelboom and Stewart (1982) argue, observed effects may result from modulation of afferent *or* efferent arms of physiological systems regulated by the CNS. It should be noted that activation of an efferent neural system is likely to be monitored by an afferent system providing feedback to the CNS concerning the consequences of drug-induced effector activation. Because a major goal of the CNS is regulation, this feedback is likely to initiate CNS output that adjusts or compensates for the drug-induced effector activation.

Unfortunately, with a drug, it is often impossible to determine whether it

is producing direct afferent stimulation, or indirect afferent stimulation associated with effector systems. The complexity of such an analysis becomes even more daunting when one realizes that particular drug responses may be subserved by distinct, yet interactive, physiological systems, and each of these may be differentially affected by drug. For example, drug-induced pyretic changes may reflect central behavioral heat gain/loss systems, central physiological heat gain/loss systems, or peripheral heat gain/loss effectors. Once this difficulty is appreciated, it becomes apparent that the behavioral responses of a drug may result from either afferent or direct efferent stimulation of diverse physiological or behavioral control systems. Clearly, specification of the drug US is a difficult enterprise. It is no wonder that investigators have typically not addressed this thorny issue.

3.4 THE WITHDRAWAL US

In the alcohol-dependent organism, cessation of alcohol induces a withdrawal syndrome that includes shakes, restlessness, insomnia, lethargy, nausea, muscular fasciculations, and activation of the autonomic nervous system (ANS) and convulsions. Alcoholics sometimes report strong cravings for alcohol during withdrawal. The absence of alcohol serves as the US, whereas withdrawal symptoms are the URs and may be conditioned to withdrawal-associated cues. Because the ANS may be activated by events unrelated to withdrawal (e.g., stress), investigators should be careful in using ANS measures alone as indices of conditioned withdrawal.

This concludes our discussion of general methodological considerations in Pavlovian conditioning with the alcohol US. Additional methodological issues are taken up in the context of the three specific research domains we shall present, on preferences/aversions (§4.1), tolerance (§4.2), and alcohol craving/urges (§4.3).

4. RESEARCH FINDINGS

4.1 CONDITIONED PREFERENCES AND AVERSIONS

4.1.1 *Pavlovian Mechanisms of Food Selection*

A voluminous literature suggests that many feeding habits are acquired by processes conforming to a Pavlovian conditioning framework (e.g., see Riley & Clarke, 1977). It has been argued that preferences or aversions for the flavor of food (CS) are adjusted by the post-ingestional consequences of the meal (US) (Sherman et al., 1984a). Repletion of nutritive deficits or recuperation from illness results in a CR described as a conditioned taste preference (CTP), whereas toxicosis or nausea results in a conditioned taste aversion (CTA). In this way, the association of tastes with internal events of homeostatic significance alters the tendency of organisms to select subsequently those flavors for ingestion. Because alcohol is consumed like a food, its flavor components may be readily associated with its post-ingestional consequences. This raises

the possibility that Pavlovian principles of food selection may be of use in understanding alcohol consumption in humans.

We assume that these Pavlovian principles are applicable to both "normal" and excessive drinking habits, although it is likely that the alcohol US is different for the two classes of drinkers. For example, in the alcoholic there may be genetic and/or acquired predispositions that enhance the affective significance of the alcohol US. Or, as a consequence of excessive drinking, the development of tolerance, dependence, or brain dysfunction may alter the significance of the alcohol US. To the extent that the alcohol US is different for normal and excessive drinkers we expect the consequences of conditioning with alcohol to be different. Although the outcome of conditioning may be different, the difference presumably reflects the significance of the US, not the conditioning process — unless, of course, alcoholism has produced organic brain pathology sufficient to generally alter associative learning and/or production of the CR.

4.1.2 Generalizability of Preference Research

Most of the basic behavioral information regarding both general food selection habits and those specific to alcohol comes from conditioning studies with the laboratory rat (e.g., Barker et al., 1977; Milgram et al., 1977; Sherman et al., 1984a). Although there is evidence that the general principles of conditioned food habits derived from study of the rat are applicable across a vast phylogenetic array extending from mollusks (Gelperin, 1975) to humans (e.g., Baker & Cannon, 1979; Bernstein, 1978), the rat differs from humans in two important ways we view as specifically relevant to alcohol consumption. First, in contrast to humans the rat lacks the musculature for emesis. The rat cannot expel toxic foods that in other species would activate what is called the "emetic" centre (Borison & Wang, 1953). We would argue that the inability to expel toxic foods biases the rat to be extremely sensitive to the toxic components of post-ingestional USs, especially with regard to potential association with taste stimuli (Norgren & Pfaffman, 1975). The rat's feeding behavior reflects this biasing: It ingests small portions of novel foods and waits until the new morsel's "safety" has been established. If the new food is followed by malaise, a CTA is established. In the rat such learning can occur in a single trial (Brackbill & Brookshire, 1971; Garcia et al., 1967) and with delays between a flavor CS and toxic US of 6 hours or more (Garcia et al., 1966; Smith & Roll, 1967). Clearly, the rat is an ideal species for studying CTAs.

There is also evidence that the rat can acquire a CTP for a taste accompanying a positive change in internal state, such as recovery from illness or restoration of a caloric deficit (see Sherman et al., 1983; Zahoric, 1977). However, in the absence of emesis a bias in favor of developing CTAs makes good adaptive sense. Hence, with alcohol, a US that has a constellation of post-ingestional consequences including caloric repletion, mood alteration, and toxicosis, the rat may be biased to associate gustatory cues with the toxicity of the drug. Thus taste-mediated learning in the rat may lead to overestimation of the importance of CTAs to alcohol consumption in humans but underestimation of the significance of CTPs. Indeed, the rat displays CTAs to flavor CSs paired with common abused substances such

as morphine, amphetamine, and cocaine (e.g., Cappell & LeBlanc, 1977).

The place conditioning paradigm may provide a more useful analogue of human alcohol preferences than the taste conditioning paradigm. In this procedure a distinctive location is paired with a drug US, and conditioned effects are typically measured by the time spent in the US-associated location compared to a control location. Similar to the aversion developed to tastes paired with lithium chloride, an emetic agent, a conditioned place aversion (CPA) is shown to a place CS associated with the sequelae of lithium (e.g., Best et al., 1973); however, a conditioned place preference (CPP) is found with morphine, amphetamine, or cocaine (e.g., Mucha et al., 1982; Sherman et al., 1980a,b), clearly reflecting the status of these drugs with respect to their addiction potential in humans. Thus, the place conditioning procedure may be a useful tool for understanding conditioned preferences/aversions with alcohol.

A second factor possibly limiting the degree of extrapolation from rat to human is that, for the rat, food selection is mediated by palatability, that is, the hedonic value of the flavor (Garcia et al., 1974, 1977), whereas flavor palatability does not exclusively mediate food selection in humans. For example, increased consumption among alcoholics and heavy drinkers is associated with greater expectations of the tension-reducing properties of the drug compared to the expectations of light or moderate drinkers (Brown et al., 1985). Such cognitive expectations may underlie the excessive use of alcoholic beverages that drinkers often admit taste "bad." Thus, among humans, mechanisms other than palatability changes resulting from conditioning may importantly influence consumption.

Having given consideration to two possible limitations of information gleaned from the laboratory rat, we will review the characteristic research methods involved in the study of both conditioned taste and place effects with alcohol USs.

4.1.3 *Research Methods in the Study of Preferences and Aversions*

4.1.3a General considerations. When a rat (or human) is allowed to consume alcohol, the flavor (CS) and the post-ingestional effects of alcohol (US) are unavoidably paired. This makes any interpretation in Pavlovian terms difficult. Because our interest here is in the US properties of alcohol, the use of an arbitrary-flavor CS is of strategic importance in disentangling the influences of alcohol's flavor and effects. By permitting consumption of a flavored solution such as saccharine for a fixed period followed by the administration of alcohol in a way that blocks gustatory stimulation — for example, intragastric intubation (i.g.), intravenous (i.v.) or i.p. injection — the post-ingestional effects of alcohol may be isolated. In this way, the influence of the alcohol US on the effects of an arbitrary CS is observed, and the motivation to drink for effect may be assessed independently of alcohol flavor. A possible problem with these alcohol administration procedures is that their temporal and dose dynamics are clearly different from those of oral self-administration; doubts are thus raised about the comparability of alcohol administered in these ways to oral consumption. Similarly, in the place conditioning paradigm alcohol is typically administered by i.g., i.p., or i.v. routes. Since comparable conditioned effects

are obtained within either taste or place conditioning paradigms whether the alcohol US is orally self-administered or administered by non-oral routes (e.g., Eckardt, 1975; Lester et al., 1970; Sherman et al., 1983; Stewart & Grupp, 1986), these concerns may be unimportant.

The associative nature of the effects obtained in the taste aversion and preference conditioning paradigms has been satisfactorily established (cf. Revusky, 1977; Zahorik, 1977). Fewer studies have been aimed at eliminating non-associative accounts of conditioned place preferences and aversions, although the use of the discriminative conditioning paradigm and unpaired control procedure has implicated a role for associative processes in place conditioning (e.g., Cunningham, 1979; Sherman et al., 1983). A recent study with morphine suggests that conditioned place preferences sometimes may be due to non-associative habituation processes that are altered by the drug (Scoles & Siegel, 1986). However, its relevance to alcohol place conditioning has yet to be established, and even the generality of this concern with the morphine US may be limited (Advokat, 1985; Katz & Gormezano, 1979; Mucha & Iversen, 1984). Moreover, as we will see, the majority of place conditioning effects with alcohol also make this concern less relevant.

4.1.3b Specific methods. In the typical experiment on conditioned preferences/aversions, a rat is exposed for several trials to a CS paired with alcohol, and change in the rat's response to the CS is assessed. A conditioned preference (CP) is evidenced by increased approach or contact with the CS as a consequence of the CS-US association, whereas a conditioned aversion (CA) is evidenced by increased withdrawal from the CS. The specific measure of conditioning will depend on the nature of the CS.

As mentioned above, taste and place cues are the two classes of CS that have been studied most extensively. With a taste CS, the measure of conditioning is a change in the amount of consumption of the taste. A typical taste conditioning experiment is conducted in the following manner. A thirsty, or hungry, rat is presented with a distinctly flavored solution (e.g., saccharine) for a limited period. Following consumption of the taste CS, the rat is administered the alcohol US via i.p. injection or gastric intubation. At the next experimental session (usually 24–48 hours later), under the same experimental conditions the rat is again presented the taste CS. If an increase in consumption is evidenced relative to non-associative controls (e.g., unpaired, or randomly presented CS and US), a conditioned preference has been obtained. Alternatively, if consumption decreases, an aversion has developed.

For place stimuli, conditioned preferences or aversions have been typically indexed as an increase or decrease in the amount of time spent in an alcohol-associated environment under choice conditions. For example, with the rat a distinctive place in the test apparatus constitutes the CS. In testing, the rat is given a choice between the alcohol-paired place and either a novel place or one that is familiar to the rat but has not been paired with alcohol. A conditioned place preference is evidenced by an increase in the time spent in the alcohol-associated place, relative to controls, and a conditioned place aversion is indexed by a decrease in the time spent there.

4.1.4 Review of Literature on Alcohol-Conditioned Preferences and Aversions

A conditioned preference or aversion suggests that the rat has come to associate either aversive or rewarding effects of the US with the CS. The taste CS is of special interest with the alcohol US because alcohol is typically consumed orally and has a potent gustatory component. The place CS is of special significance because it seems to be more sensitive to the dependence-related psychoactive effects of drugs than the taste CS.

In this section we will first discuss the effects of alcohol in establishing conditioned preferences or aversions with taste and place CSs. These treatments will be divided further in terms of whether rats were drug naive or experienced prior to conditioning.

4.1.4a Taste CSs — drug naive rats. The results of numerous experiments show that taste CSs paired with alcohol yield conditioned taste aversions (CTAs) in drug-naive rats (e.g., Berman et al., 1974; Cappell et al., 1973; Cunningham, 1979; Eckardt, 1975; Kiefer et al., 1980; Lester et al., 1970). This result has been obtained with varied taste CSs, doses (e.g., Berman et al., 1974; Cappell et al., 1973), and routes of US administration including oral self-administration (e.g., Eckardt, 1975), i.g., i.p., and intracardiac (e.g., Lester et al., 1970). Thus, it appears that the effect of alcohol in yielding CTAs is robust. Alcohol is not unique in this regard, as many other drugs of abuse are effective in yielding CTAs (Cappell & LeBlanc, 1977).

One study provides some evidence suggesting which component of the alcohol US produces the CTA. Kiefer et al. (1980) tested the possibility that gastric or intestinal irritation mediates or contributes to the alcohol CTA. Rats with subdiaphragmatic vagotomies were compared to sham-operated rats in the acquisition and extinction of an alcohol-induced aversion. The vagotomized rats developed an aversion equivalent to those of sham-operated rats whether or not alcohol was administered i.g. or i.p. Because vagotomy eliminates the majority of afferent input from the gastrointestinal tract to the brain, the blood-borne effects of alcohol are apparently sufficient to support aversion acquisition.

In a recent study designed to explore the possibility that a conditioned taste preference might be obtained, Sherman et al. (1983) simultaneously studied conditioned taste and place effects with hungry rats. In their procedure, on some days rats were placed in one distinctive compartment of a two-compartment shuttlebox with a distinctive taste present. On other days, the rats were placed in the other compartment with a different taste present. One side of the shuttlebox and one taste were consistently paired with alcohol (CS+) and the other side and flavor with the vehicle control (CS-). Fluid consumption was motivated by food deprivation rather than fluid deprivation, in contrast to the above experiments in which CTAs were obtained with alcohol. Because the distinctive taste cues were embedded in a 5% sucrose solution, the hungry rats avidly consumed the taste.

In their first experiment, rats received one of three doses of ethanol (0.5, 1.0, or 2.0 g/kg i.g.). In choice tests conducted in their homecages, rats

demonstrated a CTA following the 2.0 g/kg dose, no effect at the 1.0 g/kg dose, and, of particular interest, a taste preference at the 0.5 g/kg dose. To explore the possibility that caloric restoration accounted for their results, in a second experiment they found that an isocaloric glucose solution yielded the same magnitude of preference as the low ethanol dose. Moreover, when the low ethanol dose was compared to an isocaloric glucose solution rather than the water vehicle, the alcohol-associated taste was less preferred than the glucose-associated taste. These results suggest that ethanol's caloric effect, rather than its pharmacological action, supported the conditioning of a flavor preference. (For further support of this conclusion see Mehiel & Bolles, 1984, and Fedor-chak & Bolles, 1987.)

The fact that the highest dose of ethanol used in their first experiment conditioned a flavor aversion indicates that higher doses produce aversive consequences that outweigh the benefits of caloric restoration. In fact, the second experiment suggests that even at the preferred low dose, when the caloric advantage is cancelled by the isocaloric control solution, aversive effects of alcohol may be unmasked. These findings speak to the multiple effects of the alcohol US and the challenge of determining the component(s) yielding a particular consequence.

In a follow-up study, Deems et al. (1986) explicitly manipulated the deprivational state of rats to further test the caloric restoration hypothesis. In this study rats were conditioned in their individual homecage and only taste cues served as the CS+ and CS- for alcohol and vehicle, respectively. Food-deprived rats, but not water-deprived rats, displayed a CTP for the alcohol-associated taste. The water-deprived rats tended to show an aversion for the alcohol-associated taste, but this effect was small.

Taken together, these results suggest that the CTP observed with a low dose of alcohol was due to the calories provided. However, at a high dose the aversive effects of the drug clearly outweighed its caloric effect. The results do suggest that it is possible that the taste of alcohol itself could become preferred via its caloric effects. This hypothesis has been offered recently as one alternative to account for the increase in the oral consumption of alcohol observed in hungry rats (Stewart & Grupp, 1985). Although several theories of alcoholism based on the nutritive effect of the drug have been criticized (see Wallgren et al., 1970), caloric factors may nonetheless play an important role in the early stages of alcoholic drinking habits.

4.1.4b Taste CSs — drug pre-exposed rats. Alcohol pre-exposure has been used in conditioning studies for numerous reasons including the opportunity to study how tolerance or physical dependence influences the reinforcing effects of the drug. Several studies show that if rats are pre-exposed to alcohol, the strength of the CTA is attenuated (Barker & Johns, 1978; Berman & Cannon, 1974; Cannon et al., 1975, 1977; Randich & LoLordo, 1979). While these findings suggest that tolerance may account for the attenuated aversion, this explanation is incomplete. It cannot account completely for results showing that (a) pre-exposure to one drug can disrupt aversion conditioning with a pharmacologically dissimilar agent (Cannon et al., 1977) and even with a nonpharmacological agent (Braveman, 1975), or (b) disruption results even

when animals receiving drug pre-exposure show more intense illness during conditioning than non-pre-exposed animals (Cannon et al., 1975). However, Cannon et al. (1977) demonstrated that disruption tends to be greatest when the same drug is used during both pre-exposure and conditioning, evidence suggesting that tolerance may account for a portion of the pre-exposure effect. The balance of the effect may be attributable to associative mechanisms related to the contingency analyses of conditioning presented earlier (cf. Cannon et al., 1977; LeBlanc et al., 1978). Simply, if the alcohol US is already predicted by other extra-experimental stimuli or if the contingency between the CS and US is degraded, attenuated conditioning would be expected. (See Hinson, 1982, and Randich & Haggard, 1983, for accounts of US pre-exposure effects using nonpharmacologic USs.)

Numerous experimenters have induced physical dependence by pre-exposure to alcohol, in the hope of establishing a CTP for a taste paired with alcohol-induced relief from withdrawal (e.g., Begleiter, 1975; Crawford & Baker, 1982; Deutsch & Koopsmán, 1973; Marfaing-Jallat & LeMagnen, 1979; Trapold & Sullivan, 1979). This approach has been of interest because the alcohol withdrawal syndrome is ameliorated by alcohol intake, and withdrawal-induced craving is a central construct in many models of alcoholism (Jellinek, 1960). In rats, once such physical dependence is established, abstinence induces a withdrawal syndrome that shows a time-course like that observed in humans. The development of CTAs to tastes paired with the syndrome (e.g., Crawford & Baker, 1982) suggests that withdrawal is aversive to the rat.

The evidence is at best equivocal that a CTP is established by pairing a taste CS with alcohol administration to the rat in withdrawal. Most of the studies have not yielded a CTP (e.g., Begleiter, 1975; Crawford & Baker, 1982; Marfaing-Jallet & LeMagnen, 1979); and the two studies showing a CTP (Deutsch & Walton, 1977; LeMagnen et al., 1980) suffer from methodological problems that complicate their interpretation (cf. Baker & Cannon, 1982).

The administration of a small dose of alcohol during withdrawal does appear to protect against the development of a CTA that would otherwise occur (Crawford & Baker, 1982; Marfaing-Jallet & LeMagnen, 1979). This finding suggests that alcohol does have some ameliorative effects during withdrawal that can be detected with the taste conditioning paradigm but only to the extent of blocking a CTA; the establishment of a clear and reliable CTP in the context of withdrawal remains to be demonstrated. Hence, if withdrawal-relief is important in producing alcohol preference, the CTP in the rat does not model this motivational process well.

4.1.4c Place CSs — drug naive rats. In rats that have not been exposed to alcohol prior to conditioning, the majority of studies show that rats display a conditioned aversion to a place paired with alcohol (Cunningham, 1979, 1981; Sherman et al., 1983; Stewart & Grupp, 1985, 1986; van der Kooy et al., 1983). The production of a conditioned place aversion (CPA) with alcohol has been shown to be dose dependent (Sherman et al., 1983; Stewart & Grupp, 1985; van der Kooy et al., 1983), and obtained with several routes of administration including oral self-administration (e.g., Stewart & Grupp, 1986), i.p. (e.g.,

Cunningham, 1979), i.g. (e.g., Sherman et al., 1983), and i.v. (van der Kooy et al., 1983), and under conditions of food deprivation (Sherman et al., 1982; Stewart & Grupp, 1985), water deprivation (Cunningham, 1979), and no deprivation (Cunningham, 1979; van der Kooy et al., 1983). It appears that doses of ethanol greater than 1 g/kg are needed to demonstrate a CPA (see Asin et al., 1985; van der Kooy et al., 1983).

However, despite the fact that alcohol CPAs seem quite robust, evidence of CPPs has been reported by Black et al. (1973) and Stewart & Grupp (1985). Systematic attempts by Cunningham (1981) to replicate the Black et al. findings failed to yield a CPP, and, in fact, revealed a CPA. It is not clear what accounts for their discrepant outcomes, but clearly, if truth is determined by votes, Cunningham's findings stand with the majority of findings.

In recognition of the idea that a stimulus may be either reinforcing or punishing depending on the environmental context of its administration, Stewart and Grupp (1985) explored the possibility that a conditioned preference or aversion for alcohol might be influenced by the conditions under which the drug is administered. They investigated the effects of ethanol on place conditioning under two conditions that often parallel alcohol consumption in humans: in the context of food consumption and social contact.

In their first experiment hungry rats were injected with alcohol (175, 250, 350, 500, 700, 1000, or 1400 mg/kg) and saline on alternate days, each substance paired with a distinct place CS. Half the rats in this study had access to food pellets (Food) during both alcohol and saline place conditioning trials and half did not (No Food). In a choice test, relative to no-drug controls given saline in both distinct places, the No Food treatment yielded aversions at the 1000 and 1400 mg/kg doses, whereas the lower doses yielded neither preference nor aversion — results generally consistent with those in the literature. In contrast, the Food condition produced a preference at the 500 mg/kg dose, and blocked or attenuated the aversions that otherwise occurred at higher doses in the No Food condition.

A simple hypothesis regarding the role of caloric restoration cannot satisfactorily explain their data, because alcohol provided calories in both the No Food and Food treatments, yet an alcohol CPP was only observed in the Food treatment. Moreover, the authors found that the total numbers of calories obtained in each place were generally comparable. To test the hypothesis that alcohol simply enhances the reinforcing effects of any reinforcer, including food, they tested the effects of alcohol on the CPP produced by the presence of a conspecific (a social reinforcer). In contrast to the effect of alcohol with food, a 500 mg/kg dose of alcohol diminished the CPP that would have otherwise occurred. Thus, the presence of food seemingly modulated the rewarding effects of alcohol, or alcohol specifically enhanced the rewarding effects of food. In either case, their findings should encourage further study on the role of environmental context and alcohol conditioning.

4.1.4d Place CSs — drug pre-exposed rats. We are aware of only a single study that has pre-exposed rats to alcohol in the place conditioning paradigm, that of Reid et al. (1985). Without preconditions of hunger their pre-exposure procedures yielded a CPP. In their study one group of rats was

given 1-hour access to a 6% ethanol solution followed by 3-hour access to water; the rats were otherwise fluid deprived. This procedure was in effect for 26 days and was discontinued 35 days prior to the start of conditioning. The deprivation procedure effectively produced ethanol consumption; 8–11 mL of ethanol was consumed per day. Place conditioning followed in which rats were exposed to one side of an experimental chamber paired with an i.p. injection of ethanol (1.0 g/kg) and the other side of the chamber paired with control saline injections. Following repeated conditioning trials, choice tests showed that relative to controls, rats displayed a preference for the alcohol-associated place (i.e., a CPP). It might be noted that because a 35-day interval separated the last pre-exposure session and the first alcohol conditioning trial, alcohol-induced recuperation from withdrawal could not have accounted for the obtained preference. Rather, these data suggest that pre-exposure may have yielded a long-term retention of tolerance to an otherwise aversive component of alcohol. The loss of an aversive component of the alcohol US with repeated exposures is consistent with the literature showing an attenuation of CTAs with US pre-exposure. That pre-exposure to alcohol permitted the development of a CPP is of importance because it suggests that alcohol, like other drugs of abuse, can support a CPP under conditions unrelated to its caloric effects. The finding that pre-exposure to alcohol does not clearly yield a conditioned preference with the taste CS may reflect a natural bias regarding the kinds of events that become conditioned to interoceptive and exteroceptive cues. Of interest, a recent study by Smith and Holman (1987) suggests that the different responses elicited by taste and place stimuli, rather than differences in their interoceptive and exteroceptive nature, are critical in accounting for their selective effects in conditioning preferences and aversions.

4.1.5 *Summary and Implications*

In the above review, for almost every study in which a taste CS was paired in a Pavlovian fashion with the administration of an alcohol US, a conditioned taste aversion was obtained. The notably few exceptions were those cases in which (a) a low dose of alcohol provided caloric restoration to food-deprived rats (Deems et al., 1986; Sherman et al., 1983) and (b) alcohol may have acted to diminish the pain of withdrawal (i.e., when alcohol had a medicine effect) (Deutsch & Walton, 1977; LeMagnen et al., 1980). In the latter case, however, this result has not been replicated, and it has been argued that methodological problems make interpretation of the alleged medicine effect difficult (Baker & Cannon, 1982).

Thus, evidence gleaned from the laboratory rat in the taste conditioning paradigm suggests that the caloric effect of alcohol may contribute to the acquisition and maintenance of alcohol consumption by establishing a CTP. Note that this effect was only found with a low dose of alcohol, one that yielded little evidence of intoxication. However, it is unlikely that the caloric consequences of alcohol can account for any major share of the variance contributing to alcoholism via conditioning mechanisms. Clearly, hunger has not been reported as a reason for drinking among heavy drinkers or alcoholics (e.g., Ludwig & Stark, 1974; Mathew et al., 1979), nor is it likely that an isocaloric glucose solution would serve as a substitute for a drink. Overall, the reviewed

literature suggests that alcohol is more likely to *limit* consumption of alcohol by conditioning an aversion to associated tastes than promote consumption by the conditioning of a preference.

We should now ask whether this general conclusion is due to the rat's unique adaptive predilection to associate taste cues with the aversive components of the alcohol US rather than potentially positive components. The answer seems to be no. For the most part, even when the place conditioning paradigm is used with the rat, alcohol generally conditions an aversion. This contrasts with the results obtained with other drugs of abuse such as morphine, heroin, cocaine, and amphetamine — all of which readily condition a place preference while similarly yielding an aversion to an associated taste CS. However, at least two studies reviewed above encourage further study of the alcohol CPP (Reid et al., 1985; Stewart & Grupp, 1985). The suitability of the rat as a model for the understanding of the role of conditioned preference effects needs further consideration. The preponderance of data suggest that the rat is not the species of choice in studying conditioned preferences for alcohol.

We are left with a substantive literature indicating that Pavlovian processes *clearly* contribute to the development of aversions for tastes associated with alcohol. Can these data be of use in understanding drinking patterns among alcohol drinkers, including alcoholics? Perhaps yes. First, both interview and experimental evidence confirms that conditioned taste aversions may be acquired in humans (e.g., Baker & Cannon, 1979; Cannon & Baker, 1981; Lamon et al., 1977), and specifically, the taste of alcohol beverages may serve as the taste CS (e.g., Baker & Cannon, 1979; Logue et al., 1981). With regard to the role of the alcohol US, in an interview study by Logue et al., about 25% of 517 respondents reported alcohol-related taste aversions. These aversions were usually acquired at about the subjects' legal drinking age — presumably before the skill of titrating dose had been cultivated. Even among alcoholics, there is evidence that alcohol can condition taste aversions. A recent questionnaire study of 70 hospitalized alcoholics revealed that 45% had strong aversions to the flavor of a specific alcoholic beverage (Smith et al., in preparation). Most of these aversions developed in the patients' early adolescence, or early in the course of heavy alcohol consumption when overconsumption was followed by malaise. Many of these aversions persisted over decades, and patients claimed they never drank the beverage even if it was the only alcoholic beverage available during withdrawal. These data clearly suggest that humans, like the laboratory rat, are also subject to the development of alcohol-induced taste aversions. Thus, avoidance of some alcoholic beverages may be determined by Pavlovian food selection processes; unfortunately, alcoholics drink alcohol despite its tendency to condition aversions to associated tastes.

In view of the miseries resulting from overconsumption, it is of interest to consider why alcoholics do not form more pervasive aversions to alcoholic beverages. Conditioning principles may provide a partial answer. First, if initial exposure to alcohol does not result in overconsumption and consequent toxicosis, the drinker is essentially given an exposure to the alcohol flavor CS

and an attenuated version of the potential toxic US. It is known that either CS or US pre-exposure attenuates the subsequent effects of conditioning trials (see §2.4). The CS pre-exposure effect has been clearly shown in taste aversion learning (e.g., Domjan, 1972), and the literature on alcohol pre-exposure reviewed above is consistent with this generalization about US pre-exposure. Second, recall that rats given a dose of alcohol capable of inducing an aversion failed to display a CTA if conditioning was conducted during withdrawal from alcohol (e.g., Crawford & Baker, 1982). It may be that the well-known "cure" for hangover, the "hair of the dog that bit you," may serve to attenuate the development of aversions among alcoholics. Additional considerations not directly related to Pavlovian principles include the possibility that if aversions do not develop before the alcohol US has become tolerated, the subsequent impact of alcohol's aversive effects may be too weak to support conditioning of aversions. Finally, alcoholics have a vast array of flavors that may be used as vehicles for consumption. If one becomes aversive as a result of conditioning factors, alternative vehicles abound.

The fact that taste aversions can be established in the laboratory with rats has been used as an impetus for the treatment of alcoholism with Pavlovian procedures. Results with alcoholics clearly show that pairing an emetic US with the taste CS of alcohol results in decreased consumption, decreased affective ratings of the taste, and increased heart rate responses to the taste (Cannon & Baker, 1981; Cannon et al., 1986).

These indices of CR magnitude have been found to predict duration of posttreatment abstinence (Cannon et al., 1981, 1986). Indeed, CR magnitude accounted for up to 25%–30% of variance in latency to first drink among those relapsing (Cannon et al., 1986)! Interestingly, cognitive factors seem relatively unimportant: aversions are maintained even though treated patients were aware that the taste did not cause the illness (Cannon & Baker, 1981).

In comparing the effects of emetic therapy with shock therapy, Cannon and Baker (1981) found that only the emetic treatment produced an aversion to alcohol — a result consistent with the bias of rats to associate taste cues with illness rather than painful peripheral insult. A follow-up study found that emetic therapy subjects showed a greater number of abstinent days than control and shock therapy subjects at a 6-month period; at 1 year the emetic and control groups did not differ, but both were abstinent a greater number of days than the shock group (Cannon et al., 1981). Clearly, a Pavlovian framework can be useful not only in understanding the patterns of drinking among alcoholics (beverage selection), but also in understanding which classes of behavior therapy may be most effective (emetic vs. shock) and, among subjects receiving emetic therapy, predicting success of treatment (CR magnitude).

Before we end this section, it might be useful to consider whether CTPs influence alcohol consumption in humans; clearly, the evidence is pithy in rats. We have all witnessed the sigh of relief and expression of "That's good!" in the apparently stressed drinker and/or alcoholic immediately upon tasting the "needed" alcohol beverage. Such drug anticipatory reactions seem to occur too soon to be attributable to the pharmacological effect of the drug. It seems that the anticipated relief from stress is a clearly welcome reaction and may be

a conditioned response to the alcohol taste. From this perspective, taste conditioning paradigms may not have yet fully explored the appropriate procedures for studying CTPs with alcohol. Perhaps if in a stress situation a taste (or place) CS were repeatedly paired with alcohol's sedative (stress-relieving) properties, a CTP might be obtained. However, anecdotal evidence is flexible. We note the occasions when a stiff drink is swallowed with a scrunched-up face, accompanied by shivers and raised shoulders, and followed by a less potent "chaser" to clear the palate. It seems, in such cases, that the desire to drink for the alcohol effect outweighs the aversive signals coming from the tongue via conditioned or unconditioned processes. Whereas palatability seems to sway consumption in the rat, humans seem to have the ability to override that system to some degree. The rat will also learn to self-administer alcohol to the point of intoxication, but care must be taken to circumvent the wisdom of its palate (Numan & Gilroy, 1978). That is, non-oral routes of administration must be used.

4.2 ALCOHOL TOLERANCE

Tolerance is operationally defined either as a decrease in the effect of a drug with repeated administrations, or as the need for increasingly larger doses to produce the same initial effect. As noted above, alcohol has many different actions, and tolerance may develop to these at different rates, or develop to some and not to others. For example, tolerance has been demonstrated to the hypothermic (Lé et al., 1979; Mansfield & Cunningham, 1980), psychomotor (Mansfield et al., 1983; Wenger et al., 1981), tail-flick reflex inhibiting (Jorgensen et al., 1985), and hypnotic (Melchior & Tabakoff, 1981) effects of alcohol in rats and mice; on the other hand, tolerance does not appear to develop to certain locomotor activating effects of alcohol in rats (Masur & Boerngen, 1980; Tabakoff & Khanna, 1982), though alternative interpretations of this finding have been advanced (Hunt & Overstreet, 1977).

Historically, theorists have regarded the development of tolerance primarily as a function of repeated drug exposure per se (e.g., Jaffe, 1975). In this view, tolerance occurs because physical systems develop countervailing responses to drug actions or become insensitive to disruptive drug actions. However, in recent years psychopharmacologists have presented a cogent challenge to the belief that tolerance reflects a physiological adaption that is automatically elicited by drug exposure (Baker & Tiffany, 1985; Siegel, 1979, 1983; Wikler, 1973).

Wikler (1973) and Siegel (1979) have proposed a classical conditioning model of tolerance in which the drug itself is the US, and its pharmacological effects on physiological systems the UR. Through their consistent association with the US, drug cues in the environment (CS) come to elicit a CR. Both Wikler and Siegel noted that drug-associated CRs are frequently counter-directional to initial drug effects: Siegel dubbed such drug-antagonistic CRs as "conditioned compensatory responses" (CCRs). According to this view, the observed response to drug in the presence of the CS is the algebraic summation of the UR and the CCR; as the CCR increases in strength with repeated conditioning trials, the observed drug effect decreases. Thus, tolerance may be

conceptualized as a consequence of the summation of the UR and the CCR.

4.2.1 *Methods for the Study of Conditioned Alcohol Tolerance*

A strong prediction of the Pavlovian model of tolerance is that tolerance should be specific to cues signalling drug administration. If the CS is important in modulating the drug UR, tolerance acquired in the presence of reliable drug administration cues should be specific to those cues. Tests of the Pavlovian model of tolerance have followed a characteristic discriminative conditioning design, typically using either rats or mice as subjects; few studies have yet examined associative alcohol tolerance in humans. Infrahuman subjects are injected with alcohol in an environmental context, made distinctive by the use of salient noise, lighting, and/or olfactory cues (the CS+). Controls are given equal exposure to the distinctive context, but the exposure is never paired with alcohol; they receive alcohol in a non-distinctive environment (typically the homecage or colony room). Experimental subjects are given saline in the non-distinctive environment. It is important that all subjects obtain equal exposure to the US and both (CS+ and CS-) discriminative conditioning environments, to avoid novelty effects that could confound interpretation (e.g., Siegel & Sdao-Jarvie, 1986). Once the conditioning phase is complete, all subjects are given a challenge dose of alcohol in the distinctive context. (See §3.2.1 for a methodological critique of potential problems with this testing procedure.) According to the Pavlovian model of tolerance, the display of tolerance should be greater in subjects for whom the distinctive environment is predictive of drug delivery.

Rate of tolerance acquisition should also be subject to associative manipulations. Pre-exposure to the CS without drug administration will retard the rate of learning, a phenomenon known as "latent inhibition" (Lubow, 1973). Learning will also be disrupted by degrading the contingency between the CS and US; as the probability of the pairing of US and CS on any given trial declines from 1.0 towards, say, 0.5, the rate of tolerance acquisition should evidence a corresponding decline.

Another prediction of a Pavlovian model is that tolerance should be subject to extinction. That is, to the extent that the association between CS and drug accounts for tolerance, uncoupling of the association via CS-alone presentations should attenuate tolerance. (However, care must be taken to ensure that stress does not accompany the extinction procedures, because stress has been shown to influence subsequent responsiveness to drugs, independent of the uncoupling of the CS-drug association; Sherman et al., 1982.) Associative tolerance should show little spontaneous diminution with the mere passage of time; rather, tolerance must be extinguished.

Additional tests are necessary to demonstrate the role of the CCR in tolerance. According to the logic of the CCR model of tolerance, the CCR may be observed directly by administering a placebo in the presence of drug CSs; the compensatory response should be revealed in the absence of the counter-vailing drug effect. For example, if rats have become tolerant to the hypothermic effect of alcohol and are then given a placebo (saline) in the drug-predictive environment, hyperthermia should be observed. As with drug testing, the placebo test should be administered to all subjects in both CS+ and CS-

environments to assess properly the contribution of associative factors.

As an alternative to the placebo test, some researchers have recently suggested that compensatory responses may be observed more readily by testing the animal with a drug having properties isodirectional to the hypothetical conditioned response (Poulos & Hinson, 1984). According to this view, tolerant animals should evidence a greater degree of response to the test drug than non-tolerant animals, due to the algebraic summation of the predicted CCR and the unconditional effects of the test drug. This methodology has not yet been applied to alcohol research, but has been used to test CCRs in response to pentobarbital (Hinson, 1982) and scopolamine (Poulos & Hinson, 1984).

4.2.2 *Review of Literature on Conditioned Alcohol Tolerance*

4.2.2a **Acquisition and specificity of alcohol tolerance.** A primary prediction of an associative model of tolerance is that greater tolerance will be displayed in the presence of cues that have reliably signalled drug administration (CS+) than in the presence of cues that have not. Such context-specific tolerance has been demonstrated for a variety of psychoactive drugs, including pentobarbital (Hinson & Siegel, 1986), amphetamine (Poulos & Hinson, 1984), and morphine (Siegel, 1975, 1977; Tiffany et al., 1983). For alcohol, most studies have examined rats' tolerance to alcohol's hypothermic effect. Lê et al. (1979) were the first to document this phenomenon in a pair of experiments that assessed tolerance via within- and between-subjects designs. In the first experiment, after nine alcohol injections in a distinctive room, rats showed less than half their original hypothermia on the ninth trial, demonstrating tolerance development. When the tenth injection was given in the homecage (never previously paired with alcohol), there was a significant reduction of tolerance from the previous trial. The next test was performed in the distinctive room, and the rats recovered the degree of tolerance displayed following the ninth injection. The results of the between-subjects experiment were similar. Compared to rats given saline in both rooms and tested with alcohol, rats given nine injections of alcohol displayed tolerance only if tested with the drug in the alcohol-associated context. This demonstration has been confirmed with procedural variations by many independent researchers (Alkana et al., 1983; Crowell et al., 1981; Greely et al., 1984; Lê et al., 1987; Mansfield & Cunningham, 1980; Melchior & Tabakoff, 1981; York & Regan, 1982). In a recent study, Lê et al. (1987) observed the development of hypothermic tolerance in rats given 2.0 or 4.0 g/kg of alcohol. However, context-specific (CS+) tolerance was observed only at the lower dose. As the authors point out, the extent of Pavlovian control of alcohol tolerance might depend on treatment dose. At doses below 2.5 g/kg tolerance appears to be strongly under associative control (Crowell et al., 1981; Mansfield & Cunningham, 1980), whereas at that dose and higher an increasing role for non-associative tolerance is evidenced (Lê et al., 1979, 1987). This finding is consistent with Baker and Tiffany's (1985) associative and non-associative model of drug tolerance. However, it is not clear to what extent this dose effect is specific to hypothermic tolerance to alcohol.

Tolerance to alcohol's thermic effects is the most widely documented, but not the only example of associative tolerance to alcohol. Melchior and Tabakoff (1981), in addition to measuring the hypothermic effect of alcohol in mice, also assessed the hypnotic effect of the drug by measuring post-injection sleep time. In a room (CS+) previously associated with alcohol injections, mice slept less than half as long as mice with the same alcohol history but tested in a different room. The latter group slept virtually as long as mice given alcohol for the first time; that is, there was a complete loss of tolerance when alcohol was given without the CS+.

We know of only one study of humans that is relevant to the above findings. With normal social drinkers, Dafters and Anderson (1982) found that subjects displayed tolerance to the tachycardic effects of alcohol after drinking alcohol in an environment previously associated with the drug. In a test of the situational specificity of tolerance, the subjects consumed alcohol in a context previously paired with placebo. Here, tolerance to alcohol's tachycardic effect was lower than in the presence of alcohol cues, though not eliminated completely. Other authors (Beirness & Vogel-Sprott, 1984; Vogel-Sprott et al., 1984) have reported learned tolerance on various psychomotor tasks with human subjects, but these studies have not used appropriate controls to assess the associative or non-associative nature of such tolerance. Shapiro and Nathan (1986) found contextually mediated tolerance in social drinkers on a complex cognitive task, along with evidence of a performance CCR in a placebo test, but the authors did not assess the potential contribution of practice with the target response while intoxicated.

In sum, evidence from both rodents and humans, with different dependent measures, clearly indicates that alcohol tolerance can be specific to the presence of alcohol-associated cues. This is not to assert that all tolerance is associative; indeed, several studies suggest a non-associative component (e.g., Dafters & Anderson, 1982; Lê et al., 1979, 1987). Rather, the associative model accounts for a significant portion of observed tolerance, in a manner that is inexplicable on the basis of non-associative theories.

4.2.2b Latent inhibition and partial reinforcement effects. As discussed in §4.2.1, the development of associative tolerance should also be influenced by procedural manipulations known to affect the rate and magnitude of CR development in Pavlovian conditioning. Two such procedures are (1) latent inhibition, the pre-exposure of a subject to the CS before CS-US pairings, and (2) partial reinforcement, wherein presentation of the CS is not always followed by a US (reinforcer). Because these manipulations slow the rate of conditioning, they should similarly influence tolerance development. To date neither prediction has been tested for alcohol. However, Siegel (1977, 1978) has tested both predictions using morphine as the US. In the latent inhibition study, a group of rats given 18 exposures to a hot plate prior to tolerance development manifested less tolerance when tested on that hot plate than did a group with an identical drug history but only one CS pre-exposure (Siegel, 1977). The exact mechanism by which latent inhibition interferes with conditioning is disputed (Mackintosh, 1983), but the observed result is in accord with an associative model of tolerance. However, Siegel's (1977) results

have also been interpreted as a possible stress-related artifact, since CS pre-exposure included exposure to the painful stimulus of the hot plate, a procedure shown to influence morphine's analgesic action (Sherman et al., 1982).

Partial reinforcement effects with morphine as the US were tested in two identical studies, one using analgesia as the dependent measure (Siegel, 1977), the other using thermic response (Siegel, 1978). In both cases, a group in which exposure to the distinctive context was always accompanied by morphine injection (continuous reinforcement, or CRF) was compared to a group for which CS exposure was followed by morphine injection only on every fourth trial during conditioning (partial reinforcement, or PRF). Both groups received the same total number of morphine injections. Consistent with an associative view, the CRF groups showed more analgesic tolerance and tolerance to the hyperthermic effect of morphine than the corresponding PRF groups. The PRF effect of morphine on temperature has since been replicated with mice as subjects by Shapiro et al. (1983).

4.2.2c Extinction of alcohol tolerance. Conditioned responses show little spontaneous decay or "forgetting" over time. Elimination of a CR usually requires an active intervention, involving the repeated presentation of the CS alone (i.e., extinction). Hence, the passage of time per se should be relatively ineffective in eliminating conditioned tolerance. In alcohol tolerance research, extinction is conducted by repeatedly exposing the subject to the distinctive environment and/or cues of drug delivery without the drug.

Three tests of extinction of alcohol tolerance have been conducted, all with rats as subjects and body temperature as the dependent measure. Crowell et al. (1981) induced tolerance in rats with 15 alcohol injections, then divided them into two groups. One group remained undisturbed for 48 days; the other received 24 saline injections in the context previously associated with alcohol. After this, all the animals were given a series of five alcohol injections to assess how much tolerance they had retained. The extinction group was significantly less tolerant than the undisturbed group, manifesting body temperatures 1.1°C lower on the average, a result in line with prediction. The fact that both groups lost some degree of tolerance over the 48-day interval (differences of 2.5° and 1.4°C for the extinction and undisturbed groups, respectively) indicates the operation of non-associative processes as well.

Mansfield and Cunningham (1980) performed a similar test, using seven alcohol injections to condition tolerance. They gave rats in the extinction group 12 saline injections in the alcohol-related context (CS+), and control rats an equal number of injections, but always in the non-alcohol environment (CS-). These control injections ensure comparable levels of stress as a consequence of the injections per se. When tested with challenge doses of alcohol, the control animals demonstrated a level of tolerance equal to that at the end of conditioning, whereas the extinction group animals were significantly less tolerant to alcohol's hypothermic effect. Similar findings were reported by Greeley et al. (1984). Thus, extinction appears to be an effective method for eliminating or reducing context-specific tolerance, as Pavlovian theory predicts.

4.2.2d Conditioned compensatory responses and alcohol tolerance.

In addition to demonstrating cue-specific tolerance, several investigations have been conducted to assess the role of CCRs in mediating associative tolerance (Hinson & Siegel, 1980). Five different studies have reported a CCR in alcohol-tolerant animals (Crowell et al., 1981; Greeley et al., 1984; Lê et al., 1979; Mansfield & Cunningham, 1980; Melchior & Tabakoff, 1981). In all of these experiments, the authors measured tolerance to alcohol-induced hypothermia. The CCR was a compensatory *hyper*thermia. Although Melchior and Tabakoff (1981) concurrently measured tolerance to the hypnotic effects of alcohol, they reported no compensatory response related to decreased sleep time.

Crowell et al. (1981) provided a robust test of the CCR in a full cross-over design wherein all conditioning groups were tested with both alcohol and saline in both the CS+ and CS-. All rats displayed a slight nonspecific hyperthermia when tested with saline, but rats given saline in an environment that signalled drug (CS+) were significantly more hyperthermic. As the classical conditioning theory predicts, this latter effect was opposite to the unconditioned effect of the drug and specific to the alcohol-predictive environment. However, the CCR was not as great in magnitude as the degree of associative tolerance displayed in the CS+ by the same animals.

A more typical, but less methodologically favored, test for CCRs involves administering placebo to tolerant subjects in only the CS+ (Lê et al., 1979; Mansfield & Cunningham, 1980; Melchior & Tabakoff, 1981). Using this design, Lê et al. (1979) produced equivalent results to those of Crowell et al.; that is, a significant CCR was found, but again it was of lesser magnitude than the degree of tolerance displayed. However, Mansfield and Cunningham (1980) reported a hyperthermic CCR that matched observed tolerance. Thus it appears that animals tolerant to alcohol-induced hypothermia reliably become hyperthermic when tested with placebo in the presence of alcohol cues, but the overall level of this hyperthermia is not always sufficient to account for all of the observed reduction in alcohol-induced hypothermia. This difference in magnitude of CCR and measured tolerance questions the notion that CCRs necessarily subserve all associative tolerance. Alternatively, the difference in magnitude may (1) reflect scaling and measurement problems, or (2) indicate that some initial interoceptive component of the drug itself contributes to the CS complex (Greeley et al., 1984) predicting later effects; therefore, placebo test trials without drug produce a smaller-than-expected CCR because the test CS is not identical to that during acquisition.

In any case, we are impressed with the consistency of the animal research supporting a role for a CCR in alcohol tolerance. However, studies of humans do not permit as clear a conclusion regarding that role. Dafters and Anderson (1982), who reported context-specific associative tolerance to alcohol's tachycardic effects, unfortunately did not conduct a placebo test in the drug-associated environment. Beirness and Vogel-Sprott (1984) conducted an explicit placebo test with a psychomotor task and reported that facilitation of un intoxicated performance (CCR) was positively related to the rate of acquisition of tolerance. However, confounds in the design of this study question whether

the response was specifically associated with alcohol cues, as would be necessary for a true CCR.

Newlin (1985a,b, 1986) has described a pattern of decreased pulse transit time, vasomotor activity, and finger temperature that he characterizes as an autonomic CCR, or antagonistic placebo response, to alcohol cues. This CCR complex was measured in adult social drinkers who were given placebo in an environment previously associated with two to four alcohol consumption trials. Their physiological profile was compared to their previous physiological profiles while intoxicated. The interpretation of these data is problematic because (a) subjects were not shown to have acquired context-specific autonomic tolerance and (b) his use of a single conditioning environment in testing the CCR leaves unresolved the alternative hypothesis that the placebo response was a general, not a CS-specific, adaptation. Given the previous drinking history of the subjects and no demonstration of situational specificity, the antagonistic placebo response may simply be a pre-experimental non-associative adaptation to alcohol consumption.

In summary, the most compelling evidence for CCRs in associative alcohol tolerance comes from several demonstrations of compensatory hyperthermia in rodents. Thus far, studies with human subjects have not convincingly demonstrated a *conditioned* compensatory response due to associative processes. Because Baker and Tiffany (1985) have presented an associative model of tolerance that does not posit a role for CCRs, but instead invokes the process of habituation, clear substantiation of a CCR seems critically important to the Pavlovian model.

4.2.3 Implications of Associative Models of Alcohol Tolerance

Whether or not the role of a CCR is established in associative tolerance, the situational specificity of the expression of alcohol tolerance may have significant implications. An alcoholic drinking in a familiar setting will be consuming drug in the presence of cues that elicit associative tolerance, and will experience generally predictable dose-related levels of intoxication. However, if drinking occurs in a novel context or one that is familiar but has never been associated with alcohol, associative tolerance to alcohol's effects will not be elicited; an equivalent amount of alcohol may result in higher levels of intoxication. An alcohol dose that would be sub-lethal in a previously alcohol-associated context may prove fatal in a context whose cues do not mobilize tolerance processes. Such an analysis has been proposed to account for overdose deaths in heroin addicts who died after administering doses of heroin that should not have been lethal (Siegel et al., 1982).

The Pavlovian model predicts that the risk of intoxication may be reduced by drinking at a familiar time, in a familiar place. An individual who is accustomed to having one or two cocktails at home before the evening meal may become uncharacteristically intoxicated if he or she has the same number of drinks in an unfamiliar bar in the afternoon. If this individual then attempts to drive, the loss of associative tolerance may have very negative consequences. If controlled drinking is advocated as a goal of treatment, then drinking in the presence of cues formerly paired with alcohol (CS+) should act to reduce intoxication and this may facilitate attainment of the goal of controlled intake.

Conversely, when drinkers seek out alcohol specifically for its sedating effects, they may be able to achieve more sedation/relaxation by drinking in a novel context. In this case, there will be a release from associative tolerance, providing a greater effect with less alcohol intake.

4.3 PAVLOVIAN PROCESSES IN THE DEVELOPMENT OF URGES TO DRINK

In our treatment of the relevance of Pavlovian learning to the development of urges, we will first discuss the construct of urges, present the Pavlovian model of urges or craving, and then describe briefly the characteristic methods used to research the elicitation of urges. (Although it is possible to distinguish urges and craving on a semantic basis — “craving” suggests a stronger more persistent phenomenological state — we do not distinguish between the two terms for the purposes of this paper.) Next, we will review data relevant to their elicitation for evidence implicating particular motivational processes and the role of Pavlovian learning. Finally, we will discuss the implications of the reviewed data and identify targets for future research.

4.3.1 *The Urge/Craving Construct*

Mello (1972) has questioned the value of studying urges, as opposed to an easily observed behavioral response (e.g., drinking behavior). She noted that if urges are merely states inferred on the basis of observed drinking or drug self-administration, there is no reason to invoke such a mental mediator. Mello’s critique is apt if one views urges as being functionally equivalent to drinking. Recently, however, researchers have begun to view urges as *affects* that have *attitudinal*, *behavioral*, and *physiological* response correlates (e.g., Baker et al., 1987; Ludwig & Stark, 1974; Rankin et al., 1979; Sherman et al., 1986; see also Lang, 1983; Lang et al., 1983). Moreover, Baker et al. (1987) have recently argued that these three urge components are coded into associative networks or schemata. Consistent with an associative network model, the number and quality of urge elicitors that impinge on a subject determine the magnitude of responding occurring across the three response systems. It is implicit in a “network” model that urges and any single urge-related response are not functionally equivalent. The advantage of analysing urges is precisely that such analysis should yield information not obtainable through observation of any single response system. In this formulation, urges need not be equivalent to drug self-administration: the addict may experience numerous urges and yet not take the drug; conversely, smokers commonly report smoking cigarettes without strong urges to do so.

Given that the analysis of urges represents a distinct source of information, why might it be an important focus of analysis? First, conceptually, the construct of an urge lies at the heart of some models of addiction. Phenomenological analysis suggests that all addictions are characterized by urges, and hence urge occurrence should be accounted for by any adequate model of addiction. Second, urges are presumed to have functional primacy over drinking behavior: in a causal model, urges can lead to drinking. If indeed urges have primacy over drinking behavior, their study is then justified. Finally, if

affects do indeed reflect the processing of information in motivational systems (e.g., Buck, 1985; Lang, 1983), then an analysis of urge response components may elucidate the nature of motivational processes subserving addiction motivation.

4.3.2 A Pavlovian model

According to a Pavlovian model of urges, various stimuli come to elicit urges (acquire incentive value) through their association with either drug or withdrawal from drug. Ostensibly, by systematically collecting information on the nature of eliciting stimuli and the nature of urge responses, it should be possible to identify the germinal alcohol effects that have important incentive/urge value for the organism. According to one Pavlovian view, drugs acquire strong incentive value because, over repeated administration, physiological and behavioral responses develop that are antagonistic to direct drug effects (the URs). These drug-antagonistic responses, which may be labelled "compensatory responses" (Siegel, 1983), "opponent-processes" (Solomon, 1977), or "counteradaptive mechanisms" (Wikler, 1980), attenuate the drug's appetitive effects and thereby require that greater amounts of drug be self-administered to attain those effects. Moreover, if such compensatory (homeostatic) responses are conditioned to drug-associated stimuli (CSs), then their elicitation might prompt drug-seeking in previously dependent and now abstinent organisms (Siegel, 1983). Inherent in this theory is the notion that drug compensatory responses are aversive since they oppose appetitive, direct drug effects. Presumably, if drug compensatory responses are critical to urge elicitation, one should observe that self-reported urges are accompanied by physiological responses antagonistic to direct drug effects.

In general, compensatory models assume that homeostatic responses constitute the principal substrates of the withdrawal syndrome. This view is consistent with the fact that many withdrawal symptoms are counterdirectional to direct drug effects. The associative elicitation of compensatory responses produces aversive withdrawal states, and because the alcoholic learns early that alcohol alleviates these states, the motivational significance of Pavlovian conditioned compensatory responses is clear. They serve as setting events signalling reinforcement availability for operant alcohol self-administration. However, not all withdrawal symptoms are antagonistic to direct drug effects (e.g., the tachycardia of alcohol withdrawal). Some motivational models emphasize that any withdrawal symptoms, whether conditioned or unconditioned, and whether antagonistic or isodirectional to direct drug effects, play an influential role in eliciting drug-seeking and craving (e.g., O'Brien, 1976). Such a model merely holds that alcohol urge measures be concordant with the components of the alcohol withdrawal syndrome: for example, tremor, increased respiration, sweating.

Another model of drug incentive processes emphasizes that drugs have potent appetitive effects and these may be sufficient, in themselves, to explain the compulsive drug ingestion that characterizes addiction. In a recent presentation of such a model, Stewart (1984; Stewart et al., 1984) argued that the appetitive effects of opiates and amphetamine cannot be attributed to drug compensatory responses that accrue over repeated drug presentations, or to drug withdrawal symptoms. She noted that some drugs such as cocaine are

self-administered at high rates upon initial access and do not require the development of drug-compensatory withdrawal processes to motivate drug use. In addition, she observed that one treatment that effectively reinstates drug self-administration among animals, for which such self-administration has been extinguished, is to administer small "priming" doses of drug (Stewart, 1984). Therefore, "relapse" to drug use can be associated with direct agonist drug effects, not with drug-antagonistic responses. Accordingly, urges should be associated with conditioned or unconditioned responses isodirectional to direct appetitive drug effects. Stewart conceptualizes direct drug actions as USs—the initial actions of drug constitute stimuli (Eikelboom & Stewart, 1982). Organisms may respond to initial drug actions in one of two ways. If initial drug actions occur on afferent CNS systems, the actions send a signal (US) to central regulatory mechanisms (as in control systems theory) and result in concordant effector/efferent changes; these efferent changes constitute the URs to drug, and their elicitation by drug cues (CSs) produces CRs. If initial drug actions occur on efferent/effect structures, they bypass central regulatory mechanisms. Therefore, they cause a departure from centrally regulated set-point values. The US in this case is the initial efferent drug action. Feedback—that initial drug effects have caused a mismatch with respect to homeostatically dependent set-point values—causes the organism to launch secondary responses (URs) to counter initial drug effects and restore homeostasis. Thus, such URs are antagonistic to the initial, efferently mediated, effects of drugs. Drug cues (CSs) would elicit conditioned responses isodirectional to URs (opposed to initial efferent drug effects). According to Stewart's control-systems model of drug conditioning, either efferent or afferent drug effects might have motivational significance for addiction. However, she and her colleagues (e.g., Stewart et al., 1984) have especially implicated conditioned and unconditioned afferent drug effects on appetitive motivational systems such as the dopaminergic ventral tegmental reward system (e.g., Kim et al., 1986; Wise & Bozarth, 1982). Activation of appetitive motivational systems should yield increased behavioral activity, increased pursuit of appetitive stimuli (consummatory responses), greater responsiveness to signals of reward, and positive affect (Stewart et al., 1984). Therefore, on the basis of Stewart's model one would expect self-report of urges to be accompanied by behavioral activation and its physiological concomitants (e.g., increased heart rate, muscle tonus, metabolic rate), positive affect, and increased pursuit of appetitive stimuli. According to Stewart these can be produced by direct actions of drug or by CSs paired with these effects. Stewart et al. (1984) do acknowledge that withdrawal can enhance the incentive value of drug. However, these authors argue that even when the addicted organism is in a state of withdrawal, it may show activation of appetitive motivational systems because withdrawal is often associated with self-administration of drug. Thus, the presence of withdrawal, or conditioned withdrawal, activates appetitive motivational systems because withdrawal constitutes a discriminative stimulus for maximum reward for drug self-administration.

One final type of motivational model of drug or alcohol incentive properties is the stress-response-dampening or stress-reduction model. This

model is based on the beleaguered but resilient notion that alcohol's appetitive effects are due to its amelioration of negative sensations or affects associated with stressors. Although there are conflicting data regarding alcohol's ability to ameliorate stress effects (Pihl & Smith, 1983), recent research suggests that alcohol may reduce stress responding (Abrams, 1983; Levenson et al., 1980; Sher & Levenson, 1983). Moreover, there is little doubt that alcoholics view stress and negative affects as being important determinants of urges and relapse (Marlatt & Gordon, 1979). A stress-reduction model would predict that stress, or signals of stress, should elicit increased urges.

According to a stress-reduction model, stress or stress effects serve as setting events for drug self-administration, much as withdrawal can serve as a setting event in the compensatory response model or in Stewart's incentive model. (Withdrawal would, no doubt, be considered a stressor according to a stress-reduction model.) Therefore, stressors (USs) or signals of stressors (CSs) should elicit unconditioned and conditioned stress responses (URs, CRs). Of course, this Pavlovian formulation is an alternative to Pavlovian urge models in which alcohol or withdrawal serve as the US. Typical stress responses might include increased heart rate, skin conductance, and muscle tonus. Obviously, many responses can carry water for more than one theory. Heart rate increases can be viewed as a withdrawal effect, a stress response, or a consequence of the activation of an appetitive motivational system. Therefore, identification of the important motivational influences on urges depends upon interpretation of multiple urge responses; for example, heart rate accelerations in the presence of great anxiety and dysphoria are unlikely to reflect activation of appetitive motivational systems.

4.3.3 Research Methods in the Study of Urges

The elicitation of self-reported urges by drug- or withdrawal-associated cues does not necessarily indicate a Pavlovian basis for the phenomenon. Although authors have frequently assumed that such responses in drug or alcohol addicts are Pavlovian CRs (e.g., O'Brien, 1976), no data conclusively support this, and indeed it seems doubtful that incontrovertible evidence can be forthcoming. We simply cannot, ethically, manipulate stimulus-drug contingencies in the course of development of addiction in humans. Therefore, as researchers, we are able to study only the consequence of some presumed associative process. We are unable to employ the various controls that would ensure mediation by a Pavlovian contingency.

Moreover, as with the study of the acquisition of preferences for alcohol cues in animals, we face difficulties extrapolating from animal research to the development of urges in humans. For instance, even if Pavlovian learning is responsible for preference acquisition by animals (e.g., Reid et al., 1985; Sherman et al., 1983), Pavlovian learning may be superseded by more complex forms of learning in humans (e.g., Razrini, 1971). Finally, there is a grey area with respect to the relationship between Pavlovian learning and more complex forms of learning (e.g., Dawson et al., 1982; Maltzman, 1979).

At present the principal objectives of Pavlovian research on urges are to determine: (a) events that elicit urges, (b) whether events eliciting urges may be conceptualized as CSs (e.g., the events share a contingent relationship with

alcohol use or withdrawal), (c) if urge-related responses conform to our notions of CRs with respect to latency, magnitude, and directionality (cf. Eikelboom & Stewart, 1982), and (d) whether urge-responses are affected by manipulations that are known to affect the magnitude of CRs (e.g., extinction).

We review two sets of data bases in our effort to examine the role of Pavlovian learning in urge formation and display. The first data set consists of retrospective self-reports in which respondents characterize the behavioral, environmental, or internal events prompting drug urges or drug self-administration. This work is susceptible to all the typical confounds of such research: for example, illusory correlations, faulty memories, idiosyncratic definitions. While such studies do not contain objective criteria against which the self-report data may be compared, the many replications conducted on diverse populations and drugs of abuse support their external validity.

The second data set comes from laboratory studies in which responses to alcohol or alcohol-related cues are assessed, typically, across three response channels: behavioral, physiological, and attitudinal. These studies are usually designed to yield data on the response correlates of urges, and the stimuli used to elicit urges are those identified by alcoholics as elicitors of urges or relapse (i.e., negative affects, stress, the taste or effects of alcohol, and withdrawal symptoms). The physiological self-report measures vary considerably across studies.

4.3.4 *Research Evidence on Elicitation of Urges*

4.3.4a Self-report. Two types of self-report data reveal information on the elicitation of urges: self-report of drinking or relapse and self-report of urge occurrence. If urges are affective states that are associated with increased likelihood of drug self-administration or increased positive evaluation of a drug's hedonic valence, then on these definitional grounds alone one would expect the elicitors of relapse also to elicit urges. Indeed, studies suggest that, in general, both urges and relapse are elicited or exacerbated by the same classes of events. The probability of urges increases in response to the presence of alcohol, exteroceptive cues previously associated with alcohol, the taste of alcohol, negative affect, physical symptoms similar to alcohol withdrawal, and, in a smaller percentage of problem drinkers, feelings of happiness or relaxation (Ludwig et al., 1977; Ludwig & Stark, 1974; Mathew et al., 1979). These are all potential drug-associated stimuli, and hence relevant to a Pavlovian analysis.

Although retrospective self-reports are susceptible to bias and distorted recall, there is some reason to accept the accuracy of the data. They are remarkably consistent across different populations, different interview techniques, and even different drugs (e.g., Shiffman, 1982). Moreover, the fact that consistent results are obtained, whether urge or relapse occurrence is the target of inquiry, increases our confidence in the generalizability of the results.

Of course, even if one accepts the premise that the listed stimuli do indeed prompt urges, there is no direct evidence that they come to do so through Pavlovian learning. It is conceivable that the urge network could be stimulated by the taste of alcohol, not because alcohol flavor has repeatedly signalled euphoria or withdrawal relief, but instead because of socially transmitted expectations about alcohol's effects. In the latter case, stimuli that

prime urge networks may have acquired informational value through routes other than CS-US pairings.

4.3.4b Laboratory data on the characteristics and correlates of urges. Because laboratory studies permit control over the presentation of stimuli and the concurrent assessment of urge responses across the three response systems, they can provide additional evidence on the elicitation of urges.

In one study, Eriksen and Gotestam (1984) examined the responses of 16 alcoholic subjects to alcohol-relevant and neutral photographic slides. Dependent measures included heart rate, subjective ratings (craving, anxiety), and behavioral observations (ratings of tension). The goal of the study was to determine if alcohol stimuli elicited withdrawal symptoms and signs in subjects. In contrast to the neutral slides, the alcohol slides elicited greater self-ratings of craving and anxiety. In addition, when subjects viewed the alcohol slides their behavior was rated as more tense. No differences were found in cardiac response to the two types of slides. Because alcoholics are typically tense and anxious during withdrawal, the authors attributed differences in response to the two types of slides to conditioned withdrawal reactions elicited by the alcohol slides. Though this attribution may be correct, other explanations clearly exist. Subjects may have experienced frighteningly strong urges, the alcohol may have made them feel guilty because of their past alcohol-related transgressions, and so on. This anxiety, completely unrelated to withdrawal effects, might have elicited urges to drink. In short, the authors' attribution of their craving results to conditioned withdrawal effects is subject to alternative explanation.

Pomerleau et al. (1983) performed a similar study, in which they compared the responses of eight alcoholics and ten controls to the smell of either their favorite type of alcohol or cedar chips. The results indicated that, in comparison to controls, alcoholics showed significantly more swallowing (which the authors inferred indexed salivation) and craving in response to the alcohol than to the cedar (Table 1). Pomerleau et al. observed that alcoholics' increased swallowing in response to alcohol might serve as a sensitive measure of alcohol's incentive value, though they acknowledge that they are unable to choose among the many potential explanations for this phenomenon.

Recently, Monti et al. (1987) replicated Pomerleau's finding while directly measuring salivation (shades of Pavlov!); the sight and smell of alcohol elicited greater salivation among alcoholics than among nonalcoholics. Moreover, in the Monti research magnitude of salivation was directly related to ratings of urges to drink both for alcoholics ($r = .45$) and nonalcoholics ($r = .32$). In addition, among alcoholics, ratings of urges to drink were highly correlated with scores on the Alcohol Dependence Scale ($r = .65$), although the correlation between salivation and alcohol dependence was modest ($r = .24$). Together, the Pomerleau and Monti studies suggest that salivation may be a sensitive index of urges/motivation to drink. However, as both sets of authors observe, the results yield no conclusive evidence about the basis of alcohol motivation.

TABLE I

Physiological Responses Associated with Urge Elicitation

**CORRELATE OF ALCOHOL CONSUMPTION, WORK
FOR ALCOHOL, URGE SELF-REPORT**

		PHYSIOLOGICAL EFFECTS OF URGE-ELICITORS ^a											
		Kaplan et al. (1983)	Kaplan et al. (1985) ^b	Ludwig et al. (1974)	Montig et al. (1987)	Rankin et al. (1979)	Stockwell et al. (1982)	Kaplan et al. (1983)	Kaplan et al. (1985)	Ludwig et al. (1974)	Montig et al. (1987)	Rankin et al. (1983)	Stockwell et al. (1982)
SCL	↑ ^b	↑						↑	↑	↑	↑	↑	
HRL	↑				↑			↑	↑	↑	↑	↑	
Respiration		↑								↑↑ ^c			
Alphawave			↑							↑↑ ^c			
Tremor				↑						↑↑ ^d		↑	↑
SBP									↓ ^e	↓			
DBP			↑							↓			
Body Temperature											↑		
Salivation								↑			↑	↑	

Note: SCL = skin conductance level, HRL = heart rate level, SBP = systolic blood pressure, DBP = diastolic pressure.

^a Urge elicitors might be alcohol itself or cues associated with alcohol (e.g., bar paraphernalia). Because alcohol directly exerts physical effects, some effects listed in this table may reflect unconditioned effects of alcohol.

^b Upward-pointing arrows indicate positive correlations; downward-pointing arrows, negative correlations.

^c A low dose of alcohol increased HRL, respiration, and alphawave activity; a high dose increased them even more.

^d In Ludwig et al. (1974) a low dose of alcohol increased tremor, or high dose decreased tremor.

^e A low dose of alcohol decreased systolic blood pressure; a high dose decreased it even more.

Kaplan and his associates completed two recent studies relevant to alcohol urges. In the first (Kaplan et al., 1983) alcoholics and nonalcoholic beer drinkers were allowed to hold, sniff, and then consume either beer or placebo beer. Later, subjects had the opportunity to perform an operant that allowed them to choose between more of the same drink they had sampled earlier or a lottery ticket. Alcoholics actually given alcohol rated their desire to drink as higher than all other subjects. However, this cannot be attributed clearly to the pharmacological effects of alcohol because desire-to-drink ratings were highly correlated with alcoholics' beliefs that they had indeed consumed alcohol.

Alcohol given to alcoholics resulted in elevations of heart rate (HRL) and skin conductance level (SCL) and both were related to indices of motivation to drink (Table 1). SCL was positively related to desire-to-drink ratings, and HRL was positively related to the choice of alcohol as a reward for operant responding.

In a subsequent study Kaplan et al. (1985) examined responses of 59 alcoholics and 26 non-problem drinkers to the sight and smell of alcohol. Alcohol was effective in eliciting greater SCLs and HRLs in alcoholics than in nonalcoholics, but only SCL was related to desire-to-drink ratings. Also, desire-to-drink ratings were significantly correlated with SCL in response to alcohol among alcoholics with histories of severe self-reported withdrawal symptoms (based on a median split). However, one caveat with respect to these findings is that desire-to-drink ratings were no higher among alcoholics than among non-problem drinkers.

The landmark research on alcohol urges has been conducted by Ludwig and his associates. Because of evidence that stressors elicit urges among alcoholics, Ludwig et al. (1977) tested whether alcoholic subjects reported greater urges after a failure experience than after a success experience. In addition, they assessed whether steady drinkers and binge drinkers differed in elicitation of urges, whether urges were associated with characteristic physiological activity, and whether alcohol-relevant stimuli elicited greater urges than neutral stimuli. The 54 alcoholic volunteers were randomly assigned to "Label" or "Nonlabel" conditions. In the Label condition subjects were tested in a setting that contained numerous stimuli associated with bars and drinking (e.g., bottles of liquor, liquor advertisements, the odor of bourbon, an audiotape of barroom sounds). In the Nonlabel condition subjects were tested in a laboratory environment that contained a beaker, shelves stocked with laboratory supplies, and so on. Dependent measures included subjective ratings of craving and arousal, operant responding for alcohol, basal skin resistance, electromyography, respiration rate, alphawave activity, and blood pressure. In the operant task 750 button pushes earned 1 ounce of alcohol. A total of 6–8 ounces of alcohol could be earned per session and consumed at the session's end.

After baseline measurement, subjects participated in either a Success or a Failure experience. In the Success experience subjects were told they were correct 75%–85% of the time when they labelled one member of a pair of proverbs as more "correct." In the Failure experience subjects were told they were wrong on 75%–85% of trials. There were no significant main effects for

the Success/Failure or the Label/Nonlabel conditions. There were, however, significant interactions between drinker type and condition. The Label condition exerted significantly greater effects on the Steady ($n = 17$) as opposed to the Binge ($n = 27$) drinkers as reflected by higher craving ratings, more work for alcohol, greater skin resistance, and declines in systolic (SBP) and diastolic blood pressure (DBP). These results show that subpopulations of alcoholics may be differentially affected by alcohol-associated drinking cues. From a Pavlovian perspective such differences would be due to differences in the number or consistency of alcohol-cue pairings, number or magnitude of withdrawal experiences, level of intoxication achieved, and so on. There are data that show a relationship between the severity of prior withdrawal and the magnitude of alcohol-elicited urges in sober alcoholics (Ludwig et al., 1974).

The authors also found some measures to be highly related to levels of operant responding for alcohol. Consistent with the notion that self-reported urges accurately reflect motivation to drink, ratings of cravings were the best predictor of operant responding for alcohol. Craving ratings were followed by increased alphawave activity, DBP, and respiration rate, and decreased SBP, all predictors of operant responding for alcohol. While subjects may have worked harder for alcohol because they craved it, it is also possible that they attributed their working to craving for alcohol. It is not possible to determine whether affective components of urges or behavioral responses enjoy temporal priority in their elicitation by drinking cues, and hence whether they are causally related.

The magnitude and completeness of a Pavlovian CR should increase as a function of the number of presented elements of a compound CS (e.g., Estes, 1979; Konorski, 1967). Thus, there should be stronger urges and a broader array of responses as the number of elicitors increases. Hence, one would predict stronger craving and greater response concordance in the Label condition than in the Nonlabel condition, and in the Failure condition than in the baseline condition. This is exactly what was found, as the set of six predictor variables (arousal and craving ratings, alpha activity, respiration, SBP, DBP) accounted for a greater proportion of variance in responding for alcohol in both the Label and Success/Failure conditions than in the Nonlabel and baseline conditions, respectively.

In an earlier study, Ludwig and his colleagues (Ludwig et al., 1974) had addressed the following hypotheses: (1) A low dose of alcohol should stimulate alcohol withdrawal symptoms more than a high dose (which would suppress withdrawal), and hence should elicit greater craving. (2) A realistic drinking setting (Label condition) should produce greater craving and greater alcohol acquisitive behavior than a neutral (Nonlabel) context. (3) Conditions producing greater craving should be associated with physiological responses associated with the alcohol withdrawal syndrome. (4) Maximal expression of craving should be produced by the joint occurrence of interoceptive (low dose) and exteroceptive urge elicitors.

The subjects were 24 detoxified alcoholics randomly assigned to either a Label or Nonlabel condition. Three dose levels were completely crossed with the label condition: 0.0, 0.6 (Low dose), and 1.2 (High dose) mL/kg 100%

EtOH. Dependent measures in this research included: craving ratings, two operant work schedules (one for alcohol and one for money), and several physiological measures.

Results showed that subjects receiving alcohol, with alcohol signals present (Label condition), reported greater craving than subjects receiving alcohol in the Nonlabel conditions. When the Label and the Nonlabel conditions were collapsed, both the High and Low doses resulted in greater craving ratings than the placebo condition. In addition, the influence of alcohol and signalling appeared to be additive. The pattern of results obtained with operant responding for alcohol was essentially the same. Administered alcohol (especially the Low dose) resulted in increased urges, as did the Label condition. Also, as was the case with urge ratings, the greatest operant responding was found when the Low dose was administered in the Label condition.

One interesting analysis concerned "relapse" to alcohol drinking. At the beginning of each session, some subjects voiced their intent not to respond for alcohol. However, some of these same subjects eventually decided to work for alcohol sometime after dose administration (placebo, Low, or High) and before the session's end. Such "relapses" tended to occur in the Label and alcohol administration conditions. This finding provides additional evidence that urge self-report serves as a valid index of a drug-acquisitive motivational state.

TABLE 2

Correlations of urge ratings and operant work for alcohol as a function of alcohol dose and label condition

	PLACEBO	LOW DOSE	HIGH DOSE
<i>Label</i>	.66	.92	.90
<i>Nonlabel</i>	.56	.43	.69

From Ludwig et al. (1974). The highest correlations between self-reports and urges were found when there was conjoint occurrence of urge elicitors.

According to a Pavlovian network model of urges, urge strength and operant responding for alcohol should be more highly intercorrelated as the number of urge elicitors increases. Table 2 reveals this to be the case. The highest correlations between craving self-report and operant responding were obtained in the Label condition when either a High or Low alcohol dose was administered. This finding suggests that the validity of urge self-reports, as predictors of other urge response elements, will vary as a function of environmental and pharmacologic factors. Again, as discussed earlier, these data suggest that concomitant elicitation of urge response elements is most likely

when multiple urge elicitors are present. Multiple cues to drink do not just increase the magnitude of one or more urge responses, but also their coherence.

Lastly, Hodgson and Rankin and their colleagues have conducted a line of research directly relevant to urges, or craving, for alcohol. In an early study, they were concerned with whether craving for alcohol is increased or elicited by alcohol per se. They administered low (8 g) and high (48 g) "priming" doses of alcohol to moderately ($n = 9$) and severely ($n = 11$) dependent alcoholics (Hodgson et al., 1979). (Subjects were aware of the doses they received, and so the study does not permit a separation of pharmacological and cognitive/expectancy effects.) Subjects were abstinent at the time of priming. Three hours after receiving the priming doses, subjects were allowed to drink additional alcohol and their self-rated desire for a drink and speed of drinking were measured. The high priming dose resulted in an increased rate of drinking among severely, but not moderately, dependent alcoholics. Moreover, the rate of drinking was positively related to desire for a drink.

Rankin et al. (1979) examined craving further in a study designed to explore the effects of alcohol withdrawal on craving. The craving for alcohol of 10 alcoholics was studied when the alcoholics were deprived of alcohol for 3 hours or 1/2 hour. The behavioral assay of craving was the alcoholics' consumption of two alcoholic drinks after the deprivation periods. A variety of mood/attitudinal and physiological measures were collected during the deprivation periods. Results indicated that the 3-hour deprivation period resulted in higher ratings of desire to drink and difficulty in resisting alcohol and faster consumption of alcohol than did the shorter deprivation period. Similarly, the longer deprivation period produced higher body temperatures and greater tremor (Table 1). Intercorrelations among measures showed that urge rating measures (i.e., desire for a drink, difficulty in resisting alcohol) were positively related to tremor and HRL (Table 1). The correlation between physiological responses and speed of alcohol consumption was not reported.

In a subsequent study, Stockwell et al. (1982) manipulated alcoholic subjects' expectations about what they were drinking, via a within-subjects' balanced placebo design. Subjects were given a beverage and were told either that it was alcoholic or that it was nonalcoholic; a subset of subjects in each instructional condition actually received alcohol, and the remaining subjects received an identical-tasting nonalcoholic beverage. Results of a drinking test administered 1 hour after priming showed that the severely dependent alcoholics were more influenced by the alcohol content of the administered beverage, whereas the moderately dependent alcoholics were more influenced by the instructional condition. That is, severely dependent alcoholics drank more quickly if they had received alcohol in the priming beverage, regardless of instructional set. In contrast, moderately dependent alcoholics drank more quickly when they had been told that the priming beverage contained alcohol and actual beverage content was relatively uninformative. Drinking test performance was correlated with self-rated desire for a drink.

Recently, Rankin et al. (1983) examined the effects of repeated exposures to the sight and smell of alcohol. Subjects were assigned to one of two

conditions: an experimental condition ($n = 6$) in which they were repeatedly exposed to alcohol (extinction) but not allowed to drink it, and a control condition ($n = 6$) in which the subjects were exposed to similar cues in imagination only, and imagined themselves resisting drinking. After the two kinds of exposure, each of the groups was exposed to an alcohol stimulus. Rate of alcohol consumption, and ratings of desire for a drink and difficulty in resisting a drink, constituted dependent measures.

Priming doses of alcohol, administered before treatment, reliably elicited increased ratings of craving for alcohol. In addition, alcohol exposure, in contrast to the imaginal control condition, resulted in significant decreases in the rate of alcohol consumption in posttreatment drinking tests. Alcohol exposure also reduced desire for and the difficulty of resisting a drink. Interestingly, the greatest reductions in urge measures were reported by control subjects when they were subsequently given the experimental treatment, a finding suggesting that the previous imaginal treatment, which had modest beneficial effects of its own, increased the efficacy of the subsequent extinction treatment.

4.3.5 Implications of Research on Urges

The data reviewed above provide some support for the hypothesis that urges are acquired and elicited through Pavlovian mechanisms. Cues regularly paired with alcohol ingestion or withdrawal reliably elicit verbal, behavioral, and physiological responses suggesting the elicitation of urges. Moreover, urge responses are more coherently expressed as the number of CS elements increases, and urges decrease in magnitude with repeated, non-reinforced exposure to putative CSs. However, we must emphasize that the involvement of Pavlovian processes in urge elicitation is entirely compatible with the involvement of non-Pavlovian mechanisms in urge elicitation. The involvement of non-Pavlovian modulation or elicitation of urges is suggested by Kaplan et al.'s (1983) finding that urge ratings (desire to drink) were highly dependent upon an alcoholic's *belief* that he had consumed alcohol (see Marlatt et al., 1973). In addition, Stockwell et al. (1982) observed that moderately dependent alcoholics' speed of drinking was a positive function of their belief that they had earlier consumed alcohol.

Given that urge correlates reflect conditioned responses to some extent, does the nature of these responses reveal information about the nature of urges? Table 1 displays the direction of correlation between the physiological effects of urge-eliciting cues and operant responding for alcohol, alcohol consumption, or self-reported urges. Theoretically, all of these physiological responses could be urge response elements. The data of Table 1 suggest that among the indices selected, the most reliable physiological correlates of urges, and the responses that most reliably reflect the presentation of urge-eliciting stimuli/events (e.g., withdrawal from alcohol, the taste or smell of alcohol) are: increased SCL, HRL, respiration, alphawave activity, tremor, and salivation. Do these responses reflect the conditioning of withdrawal responses, compensatory or opponent-process responses, stress responses, or responses isodirectional to the direct effects of alcohol?

To avoid tantalizing the reader with these diverse prospects, we assert that the physiological correlates of urges listed above do not unequivocally support any particular model of drug motivation. As noted by previous authors (e.g., Ludwig & Wikler, 1974), some of these responses are consistent with the conditioning of withdrawal responses. Alcohol withdrawal is characterized by tachycardia, increased respiration, and tremor (e.g., Knott & Beard, 1972; Ludwig et al., 1974). Yet, tachycardia and increased respiration are also direct effects of alcohol (Docter & Bernal, 1964; Docter & Perkins, 1960; Kaplan et al., 1983; Regan et al., 1965), and they are prototypic stress reactions as well. Of the three responses, only tremor seems rather unambiguously related to withdrawal. The apparent relationship between tremor and withdrawal is supported by observations that tremor is related not only to verbal reports of urges but also to dysphoric states characteristic of withdrawal (Rankin et al., 1979). Whereas tremor constitutes an archetypal withdrawal sign, increased proportion of time in alpha and lowered dominant alpha frequency appear to be direct alcohol effects (Holmberg & Martens, 1955; Lukas et al., 1986; Zilm, 1981). As Table 1 shows, increased alphawave activity was associated with urges in Ludwig's research; he and his co-workers found that events and stimuli that elicited urges (alcohol, alcohol cues) increased alpha activity, and that alpha activity was positively related to operant work for alcohol. Recently, Lukas et al. (1986) reported that alcohol-induced increase in alpha power coincided strikingly with social drinkers' self-reports of post-ingestion euphoria ($r = .95$). Alcohol withdrawal has been found to be associated with decreased alpha power and lowered dominant frequency (Begleiter & Platz, 1972; Zilm et al., 1980). Therefore, data suggest that the elicitation of a response isomorphic to a direct effect of alcohol, notably one associated with an appetitive alcohol effect, also is associated with urges/motivation to drink.

Other physiological responses associated with urges — salivation, SCL increases — do not lend themselves unambiguously to interpretation. Kaplan et al. (1985) noted that alcohol withdrawal is characterized by autonomic hyperexcitability; therefore increased SCL may reflect an elicited withdrawal response. Yet, urge-associated increases in SCL may merely reflect increased orienting — which is consistent with the fact that urges tend to be elicited by salient stimuli (e.g., the taste of alcohol) calling for attentional processing. Of course, the fact that the alcoholic orients to alcohol reveals nothing about the motivational basis of such orienting. The fact that SCL has been found to be correlated with measures of chronic alcohol consumption (Kaplan et al., 1985) or severity of dependence (Kaplan et al., 1983) is not particularly illuminating as to process. For example, increased orienting in severely dependent subjects might reflect the elicitation of withdrawal responses or the elicitation of anxiety in the alcoholic who is especially tempted to drink.

SCL increases have often been interpreted as reflecting sympathetically mediated stress reactions. And, as noted previously, some of the urge correlates are also consistent with the notion that urge response elements are isomorphic with stress responses: that is, that alcoholics label stress states as urges and that stress states precipitate drinking. However, the evidence does not thus far strongly support a stress model of urges for two reasons: (1) Many stress

responses are identical to withdrawal responses; therefore distinction between the two models is difficult. (2) Researchers have not systematically arranged stimulus conditions so that the nature of effective eliciting conditions differentially supports a withdrawal or stress model. Of course, as noted earlier, the two models are not mutually exclusive.

Increased salivation in response to alcohol cues is similarly difficult to interpret. Monti et al. (1987) note that alcohol can dehydrate the oral mucosa — thus salivation may reflect a compensatory response to this effect. On the other hand, discrete alcohol doses also elicit salivation (Martin & Langborn, 1971). Hence, salivation in response to alcohol cues may reflect a CR isodirectional to alcohol's direct effects.

The physiological correlates of alcohol urges suggest that diverse motivational processes subserve urges to drink. That the elicitation of withdrawal responses increases perceived alcohol motivation is suggested by Rankin et al.'s (1979) findings of direct relationships between tremor and length of withdrawal, between tremor and self-reported motivation to drink, and between tremor and a withdrawal-congruent affective state (anxiety; e.g., De Soto et al., 1985). The association between alphawave activity and operant work for alcohol, and the association of alphawave activity and the direct euphoric effects of alcohol, suggest that urges to drink may be reported when alcohol, or alcohol cues, elicit responses congruent with the pleasurable effects of alcohol: that is, when stimuli associatively prime, or elicit memorial processing of, direct appetitive alcohol effects. This notion is consistent with Stewart's suggestion that direct appetitive effects of opiates and stimulants may prime compulsive use of those drugs (Stewart et al., 1984).

Regardless of whether alcohol's incentive value can be attributed to its intrinsic hedonic effects, or to its ability to alleviate aversive withdrawal states, it seems likely that some urge correlates reflect the activation of stimulus or goal-seeking responses. Indeed, many of the responses associated with urges — increased respiration, tachycardia, and salivation — might be viewed as preparatory CRs that are motivated by expectation of an appetitive orosensory stimulus. Much research suggests that expectation or receipt of appetitive stimuli is associated with increased arousal and behavioral activation and its attendant physiological consequences: increased heart rate, respiration, metabolism (Fowles, 1980; Hall, 1985; Panksepp, 1986). Indeed, increased salivation is highly compatible with the notion that alcohol urges partly reflect preparatory, consummatory CRs. Seen in this light, urges may reflect in part the processing of response information that is an intrinsic (genetically determined) feature of organisms' pursuit or anticipation of potent incentives. There is no reason to doubt that such responses can be associatively elicited via Pavlovian processes.

A final observation with respect to the implications of a Pavlovian model of urges is that such a model directly suggests a method for weakening urges: extinction or cue exposure (see Rankin et al., 1983). This is one reason that it is important to characterize the motivational basis of urges. The conditions that are maximally effective for urge elicitation (CS conditions) should differ according to whether urges reflect stress responses, compensatory response

withdrawal states, activation of motivational systems that process appetitive drug effects, and so on. For example, the most effective urge elicitation, and hence extinction procedure, might be produced by presenting signals of interpersonal stress, alcohol availability, or alcohol withdrawal, depending on the motivational basis of the urge. Of course, it may be that urges have diverse motivational bases and the importance or relevance of the bases differs both within and across individuals.

5. RESEARCH NEEDS IN PAVLOVIAN MODELS OF ALCOHOL USE

We have reviewed three domains of research relevant to a Pavlovian perspective on alcoholism: taste preferences/aversions, tolerance, and urges or craving for alcohol. We would now, with broad strokes, suggest future directions for research bearing on the utility of a Pavlovian perspective.

5.1 CONDITIONED PREFERENCES AND AVERSIONS

For the rat, by far the most intensively studied species on conditioned alcohol effects, the literature indicated that alcohol generally conditioned an aversion — whether the CS was a taste or place stimulus. However, a conditioned *taste* preference (CTP) was obtained under conditions in which the rat was hungry and a low dose of alcohol was used; evidence suggested that the CTP was most likely due to caloric restoration (Mehiel & Bolles, 1984; Sherman et al., 1983). A conditioned *place* preference (CPP) was also demonstrated when the rat was hungry, food was present, and a low dose of alcohol was used, although caloric restoration may not have been a necessary condition for producing the CPP (Stewart & Grupp, 1985). One study demonstrated a CPP under conditions clearly unrelated to caloric consequence or hunger motivation. In this study conditioning was preceded by prolonged oral self-administration (Reid et al., 1985). Lastly, research suggests that alcohol may yield a CTP when the drug acts to diminish the pain of withdrawal (e.g., Deutsch & Walton, 1977), although failure to replicate and methodological concerns are significant.

These findings suggest possible avenues for the development of a Pavlovian model relevant to the pharmacologic appetitive motivational effects of alcohol in the rat. We suggest several possible directions for further research:

1. It would be of interest to determine if a CTP or CPP established under conditions in which caloric restoration served as the US might serve as a first step in the establishment of a preference based on the psychopharmacologic effect of the alcohol US. Perhaps after a conditioned preference had been established, the state of food deprivation could be gradually decreased. The alcohol dose could be gradually increased to levels producing intoxication. In short, we suggest a procedure designed to "fade out" the preference based on caloric restoration and "fade in" a preference based on the pharmacologic consequences of the alcohol US.

2. One study reviewed (Reid et al., 1985) demonstrated that prolonged pre-exposure to alcohol via self-administration resulted in a CPP when the alcohol US was injected. First, in view of failures to replicate conditioned alcohol preferences (see §4.1.4c) this intriguing finding should be replicated. That accomplished, it would then be of interest to determine the range of doses that would yield a CPP and if pre-exposure via self-administration is a necessary condition for this effect. Perhaps precise titration of alcohol effects is an important feature of the pre-exposure procedure, one only obtained by self-administration. Would *passive* pre-exposure by injection or gastric infusion produce the same results as self-administration? Alternatively, perhaps rate of administration during pre-exposure most importantly influences subsequent conditioned preferences. Perhaps a rat's receiving passive administrations of alcohol at the same rate produced by oral self-administration would set the stage for a later preference. Rats in the Reid et al. study self-administered alcohol when water-deprived; to what extent does deprivation state modulate later preference? Lastly, would a taste conditioning paradigm produce a CTP under conditions yielding the CPP, or would an associative bias produce a preference only with a place CS? The answers to these questions would greatly contribute to our understanding of the CPP obtained in the Reid et al. study. Such information might suggest the rat could be a suitable model for the study of alcohol conditioned preferences.
3. We are not aware of studies that have attempted to show the conditioning of a place preference among rats experiencing alcohol withdrawal symptoms. It may be that the amelioration of the aversive withdrawal syndrome by alcohol could produce a CPP, providing clearer evidence of the conditioned motivational significance of withdrawal alleviation than has been provided with the taste CS. Also, it would be of interest to ask whether a CS+ paired with a stressor plus alcohol would be preferred to a CS- paired with the same stressor without alcohol. The sedative properties of the alcohol US have not been studied in a conditioned preference paradigm. It may be that the amelioration of aversive events by alcohol could condition potent preferences.
4. Lastly, once a paradigm yields a CPP or CTP, it would be important to show that associative manipulations, such as extinction, partial reinforcement, latent inhibition, delay of reinforcer presentation, etc., modulate the conditioned preference in a manner consistent with expectations based on classical conditioning principles.

5.2 TOLERANCE

The basic research issue addressed in the section on tolerance was: to what extent does alcohol tolerance conform to a Pavlovian analysis? Our treatment of this subject has been based primarily on Siegel's Pavlovian model of tolerance. The basic predictions of this model are: (1) tolerance should be specific to the CS context in which drug was delivered; (2) a placebo test in the CS context should reveal a CCR (conditioned compensatory response)

opposite in direction to the initial effect of alcohol; and (3) manipulations known to modulate the strength of the CS-US association should similarly influence tolerance development and the strength of the CCR. Our review of the literature provided evidence that was often consistent with these predictions. However, we suggest that researchers:

1. Document that the CCR is specific to the presentation of the CS. This step has most often been a problem in studies involving human subjects. In order to meet the burden of proof for a CCR, the observed response must be specific to the CS context, not merely a generalized adaptation to alcohol.
2. Address why the magnitude of change observed during tolerance acquisition is typically larger than the magnitude of the observed CCR. Of the studies reviewed that assessed the magnitude of both tolerance and the CCR, only one (Mansfield & Cunningham, 1980) found equivalent levels of both. Is the discrepancy between the magnitude of tolerance and the purported CCR due to measurement problems, or might associative tolerance mechanisms that do not yield a CCR (Baker & Tiffany, 1985) adequately explain the difference?
3. Demonstrate parallel changes in tolerance and the CCR as a consequence of manipulations known to affect Pavlovian conditioning, such as extinction, partial reinforcement, etc. We are unaware of a single study using an alcohol US that has shown parallel changes in tolerance and CCRs. It would seem that such a basic demonstration is necessary support for the role of the CCR in tolerance phenomena. Data on extinction manipulations would be of particular relevance for treatment approaches.
4. Measure more than a single response system. It would be of interest to determine the generality of the influence of Pavlovian processes on tolerance. Not all response systems subject to tolerance may be influenced by Pavlovian processes; moreover, the rate of acquisition and extinction may prove to be different in different systems. To date, researchers have primarily studied tolerance to the thermic effects of alcohol.
5. Characterize the locus of the alcohol US. As we noted above, a pharmacological US can produce input to the CNS via direct afferent stimulation or via feedback from the activation of an efferent arm of a regulatory system (Eikelboom & Stewart, 1982). Depending on how the alcohol US activates the CNS, the UR may be in the same direction as the observed drug effect or in the opposite direction. Such an analysis has important implications for the characterization of the placebo CR as compensatory or isodirectional to the UR.
6. Determine whether taste cues play a role in associative tolerance. Because taste stimuli are reliable cues for the sequelae of alcohol consumption, it is of practical and theoretical importance to determine the extent to which these cues play a role in associative tolerance.

5.3 URGES/CRAVING TO DRINK

We see the following as principal needs to be addressed in future research on urges:

1. Investigators should address how the information transmitted via non-Pavlovian routes influences the magnitude or coherence of urge response elements. For example, while exposure to alcohol cues may elicit urge responses, so might the simple provision of information that alcohol is available. We acknowledge that some theoreticians might argue that such information, however transmitted, may serve as a CS and thus conform to a Pavlovian framework. However, we assume that if the CS is clearly a cognitive abstraction (such as "drug is available") that is independent of any specific stimulus previously associated with alcohol (e.g., a researcher's instructions that a flashing sign reading "QRKP" means a drink is imminent), it is best conceptualized in a cognitive framework outside the domain of classical conditioning. Investigators also should examine how verbally transmitted expectancies influence urge responses elicited by CSs (e.g., the taste of alcohol). Do expectations about the total amount of alcohol available, about future performance demands, about the likelihood of one's success/competence on a challenging task, about the availability of effective coping responses other than drinking, about urge coping skills, affect urge self-reports and related responses?

We make one recommendation regarding how researchers and theoreticians conceive of Pavlovian and non-Pavlovian influences on responses. Instead of dichotomizing urge *responses* on the basis of their Pavlovian or non-Pavlovian origins (Wilson, 1987), it might be more useful heuristically to conceive of urges via a production system model (Anderson, 1976, 1982). Thus, urge response information might be viewed as affect response information that is coded (propositionally or nonpropositionally) in memorial networks. Such information is analogous to *declarative* knowledge (Anderson, 1982) and may be retrieved via a variety of routes (e.g., a Pavlovian CS, signals of drug availability, experiencing the direct effects of alcohol), and it may have both Pavlovian and non-Pavlovian origins. However, the processing of urge/affective information may become proceduralized (automatic); processing may be influenced by if-then production rules that specify when the production is appropriate, consequences of the production, and so on. Presumably, these production rules could be influenced through Pavlovian contingencies as well as through other sorts of informational/learning processes. A chief virtue of this approach is that it keeps separate, encoded memorial information from the processes that result in acquisition and activation of the network.

2. Researchers should devise methods to activate or prime motivational systems and examine the effects of such primes on urge measures. For instance, would money or social affection prime urges to drink? Would stressors prime urges? Would the effects of such primes be directly related to the affective consequences of the primes? Not only might such information be clinically important, but it would help reveal the nature of the motivational substrates of urges.

3. Recently, Stewart and others (Stewart et al., 1984) demonstrated that priming doses of drug reinstate drug self-administration in animals. Although some research has been done on priming (e.g., Stockwell et al., 1982), there is a great need for additional research on this topic. Important questions regarding priming concern the time course of priming effects on urges, the role of expectancies on priming effects, the physiological and affective processes that index priming effects, and the role of individual differences on priming effects (e.g., Type I vs. Type II alcoholic; Cloninger, 1987).

6. BOUNDARIES OF THE THEORY

Classical conditioning principles may explain how alcohol can endow an initially neutral stimulus with (a) motivational significance relevant to consumption (conditioned preferences/aversions), (b) the ability to attenuate alcohol's effects (conditioned tolerance) and the power to elicit urges to drink; nevertheless, these principles are mute with respect to the factors that provide the context for such learning. That is, classical conditioning theory does not explain the conditions giving rise to the initial pairings of CS and US. Social factors, or expectation of drug effects, are clearly better models for characterizing the genesis of drinking. Once drinking occurs, however, the opportunity for Pavlovian learning arises.

It is also the case that classical conditioning theory does not address the manner in which an organism will respond to the elicitation of a motivational state by a conditional stimulus. For example, the conditioned elicitation of craving for alcohol probably increases the probability of alcohol consumption, but other factors determine the outcome—for example, perceived and real consequences of drinking. Thus, classically conditioned responses may set the stage for drinking by eliciting alcohol-related preparatory CRs, but actual drinking is co-determined by other motivationally significant considerations, including instrumental or operant contingencies. Lastly, it is important to note that although the domain of classical conditioning theory does not make provision for individual differences based on constitutional (e.g., genetic) factors, individual differences in conditionability due to differential CS or US exposures are explicable in Pavlovian terms and may contribute to individual differences in vulnerability to alcoholism. For example, in some ethnic groups early exposure to alcohol beverages (e.g., sweet red wine) is a cultural tradition. Such pre-exposure to taste (CS) and effect (US) of alcohol may retard their later conditionability. From a Pavlovian perspective, individual differences would be related to the experiential history with CS and US. Even though predispositional vulnerability to alcoholism may be reflected in ease of conditioning of alcohol preferences, or perhaps more pronounced conditioning of compensatory-like responses, the theory of classical conditioning does not explain such phenomena.

7. CONCLUDING REMARKS

The administration of a psychoactive drug provides the opportunity for associations to develop between the effects of the drug and concomitant stimuli. Our intent has been to evaluate the adequacy of classical conditioning as a conceptual framework for understanding how such associations play a role in a subset of phenomena contributing to alcoholism. Specifically, we examined the relevance and implications of Pavlovian principles in acquired preferences/aversions for alcohol-associated stimuli, alcohol tolerance, and urges/craving for alcohol — three phenomena we believe play an important role in the development and maintenance of excessive drinking. Our review of the literature suggests that a Pavlovian perspective contributes significantly to the understanding of these phenomena, and also suggests means for their modification (e.g., extinction).

With regard to subsequent research, it is our hope that the presentation of these three content areas in a single context will aid in fostering cross-fertilization of approaches and ideas. For example, to what extent may classically conditioned tolerance to alcohol be mediated by taste cues serving as the CS? Or, does classically conditioned tolerance play a role in the conditioning of preferences and/or aversions for alcohol-associated stimuli? Or, lastly, to what extent do CSs eliciting alcohol craving also influence tolerance to alcohol? Addressing questions that bridge these content areas may help to develop a more integrated approach to the understanding of the generality/complexity of the effects of alcohol-associated stimuli.

As for the development of treatment programs, it is our hope that clinicians will acknowledge the potential importance of Pavlovian processes and develop treatment modules aimed at extinguishing conditioned alcohol effects that may adversely influence abstinence (e.g., craving). Although strictly cognitive approaches might work at uncoupling some kinds of CS-alcohol associations, simple cue exposure may be necessary in other cases. A recovering alcoholic experiencing CS-elicited urges to drink may have to struggle with processes that may be beyond his/her conscious cognitive control. Perhaps providing the alcoholic in treatment with information about the possible contribution of classical conditioning to "spontaneously" occurring urges will help him or her cope with the trials of abstinence. Alternatively, explicit cue exposure may be a useful component of treatment. Unfortunately, we do not yet know when one kind of intervention is better suited than another. Aside from treatments based on Pavlovian considerations, assessments of alcohol taste preference, compensatory CRs, or urges/craving may prove to constitute useful outcome measures of treatment success. Typically, treatments are aimed only at abstinence. Perhaps the assessment of attitudinal, physiological, and behavioral responses to alcohol-associated stimuli may serve as a better index of treatment efficacy than abstinence alone because it may reveal areas of potential relapse vulnerability. An abstinent alcoholic with strong physiological urge components may need further treatment.

We view associative learning resulting from pairings of CSs and USs as serving important and adaptive preparatory and consummatory functions.

Unfortunately, such learning processes also may contribute to behavior that is threatening to the organism's long-term viability, especially when the US is an addictive agent such as alcohol. However, an emphasis on the general adaptive function of Pavlovian learning may help to minimize evaluative judgments about the individual and focus attention on providing treatment experiences aimed at modifying the acquired association and problematic response such as the urge to drink.

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7. ALCOHOL USE AND ABUSE: A SOCIAL LEARNING ANALYSIS

G. Terence Wilson

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Social learning theory, most recently amplified as cognitive social learning theory, provides a comprehensive analysis of the psychological principles that govern the development, maintenance, and modification of human behavior. The theory assumes that both normal and abnormal behavior can be accounted for by the same set of social learning principles. Traditional conceptions of psychopathology, such as the disease theory of alcoholism, in which there is little continuity between clinical theory and practice on the one hand and experimental psychology on the other, are rejected. Social learning theory seeks to identify the psychological determinants of behavior as well as the mechanisms by which these determinants have their effects. Most important, this approach is predicated on the assumption that the ultimate worth of a theory rests in the efficacy of the procedures it generates to produce changes in complex psychological functioning (Bandura, 1969, 1977, 1986).

1. PRINCIPAL ELEMENTS OF THE THEORY: BASIC CONCEPTS

1.1 CLASSICAL AND OPERANT CONDITIONING

Social learning theory draws heavily on the principles and procedures of classical and operant conditioning, but goes beyond these principles in emphasizing the fundamental importance of symbolic, vicarious, and self-regulatory processes in human behavior. The conceptualization of classical and operant conditioning procedures differs from traditional behavioristic accounts in reflecting a more cognitive view. Current analyses of classical conditioning have moved away from the once popular notion that what was

learned consisted of simple stimulus-response (S-R) bonds. Rather, the learning of a contingent relationship between the conditioned stimulus (CS) and the unconditioned stimulus (US) defines the conditioning process. People may be exposed to traumatic events (contiguity) but not develop phobic reactions unless a correlational or contingent relationship is formed between the situation and the traumatic event. In terms of social learning theory, these correlational relationships are seen as learned expectations. It is further assumed that this learning is cognitively mediated (Bandura, 1977; Bower, 1978). Awareness is probably not a necessary precondition for classical conditioning in people; nonetheless, awareness of the relationship between two stimuli greatly facilitates the acquisition of specific responses and hastens their extinction.

There are two major processes in Bandura's (1977) social learning analysis of how a formerly neutral stimulus comes to elicit anxiety as a function of being paired with an aversive experience. The first involves learning a predictive relationship between the two stimuli on the basis of direct experience with environmental events. The second involves a self-arousal process in which the person consciously generates anticipatory feelings of anxiety in response to an antecedent event. One of the advantages of viewing learned emotional reactions in this manner is that postulating two stimulus sources of emotional responses suggests different therapeutic interventions, as discussed later in this chapter.

Consistent with radical behaviorism or an operant conditioning approach, social learning theory holds that behavior is largely a function of response consequences. In contrast to the operant conditioning approach, however, social learning theory rejects the view that response consequences always act automatically to shape behavior in a mechanistic manner. According to social learning theory, the influence of environmental events on the acquisition and regulation of behavior is also largely determined by cognitive processes. These cognitive processes are based on prior experience and determine what environmental influences are attended to, how they are perceived, whether they will be remembered, and how they might affect future action. Social learning theory emphasizes the importance of the person's active cognitive appraisal of environmental events. Learning is greatly facilitated when people are aware of the rules and contingencies governing the consequences of their actions. By observing the consequences of behavior, the person learns what action is appropriate in what situation. By symbolic representation of anticipated future outcomes of behavior, the person helps to generate the motivation to initiate and sustain current actions (Bandura, 1977).

1.2 VICARIOUS LEARNING

The importance social learning theory attaches to *vicarious learning* (*modelling*) is consistent with its emphasis on cognitive processes. In this form of learning people acquire new knowledge and behavior by observing other people and events, without engaging in the behavior themselves and without any direct consequences to themselves. Vicarious learning may occur when people watch what others ("models") do, or when they attend to the physical

environment, to events, and to symbols such as words and pictures. The influence of vicarious learning in humans is pervasive, and this concept greatly expands the power of social learning theory to account for the complexity of human behavior.

1.3 SELF-REGULATION

Behavior is not simply determined by external consequences, whether or not they are experienced directly or observed in connection with models. A major component of social learning theory is its emphasis on the self-regulation of behavior in which the person is an agent of change. Self-regulation helps explain the indisputable fact that human behavior is usually maintained and often altered in the absence of immediate external reinforcement (Bandura, 1982b; Kanfer, 1977). In this motivational process, people make self-rewards or self-punishments conditional upon the attainment of specific standards of performance. The level of self-motivation generated by success or failure in matching self-prescribed standards will vary according to the specific performance standards, the judgmental processes involved in evaluating performance, and the nature of the incentives. The performance standards themselves are a product of prior histories of differential reinforcement and modelling influences. Figure 1 summarizes the different component processes in the self-regulation of behavior through self-managed incentives.

Dysfunctions in self-regulatory systems can explain some forms of abnormal behavior, including alcohol abuse. When people make self-reward contingent on performance standards that are too severe or unrealistic, they risk loss of self-esteem, feelings of depression, and demoralization. When self-regulation is ineffective, owing either to failure to set adequate standards of conduct or to overlooking performance that falls short of adequate standards, self-defeating and socially inappropriate behavior results. Self-evaluative reactions are particularly important in the context of alcoholism as they serve as incentives that regulate socially accepted behavior. People usually strive to avoid personal actions that would elicit negative self-evaluative consequences. However, it should be noted that a given activity does not invariably lead to the same self-evaluative reaction in all people. Such reactions are selectively "activated" or "disengaged" depending upon the person and the situation (Bandura, 1977). According to this framework negative self-evaluative reactions are most strongly activated when the causal connection between negatively sanctioned behavior and its adverse effects is clearly apparent to the person. Among the many means whereby the regulatory system of self-evaluative reactions can be disengaged is the attribution of responsibility to something or someone other than oneself. Alcohol is one such source of misattribution of personal responsibility (Wilson, 1981).

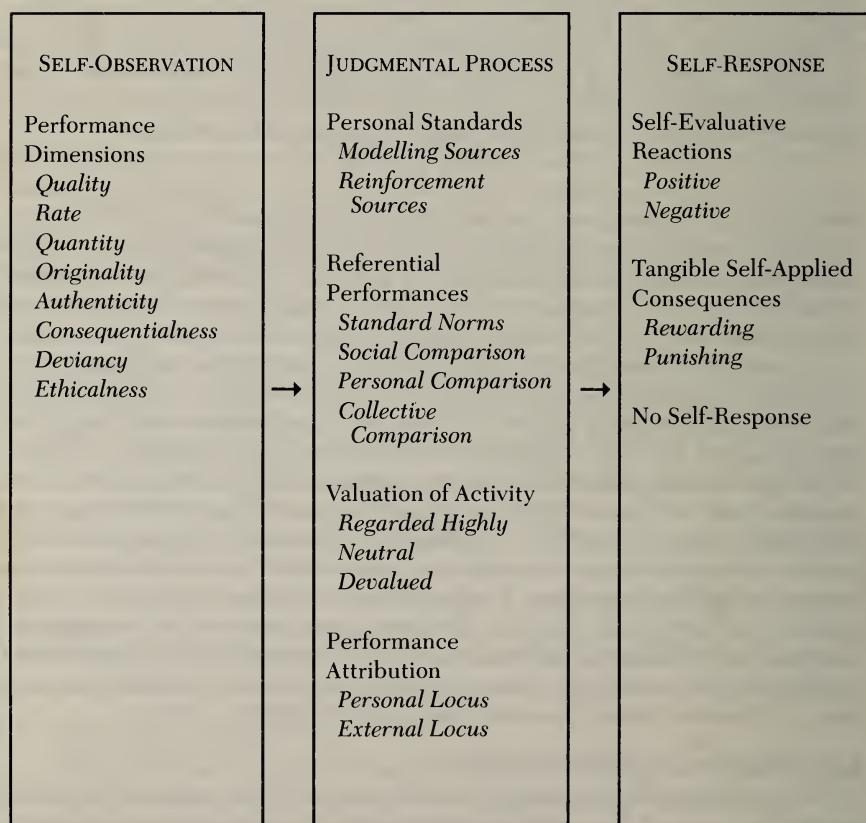
1.4 RECIPROCAL DETERMINISM

Psychological functioning involves a reciprocal interaction among three interlocking sets of factors: behavior, cognitive factors, and environmental influences. In this conceptual scheme a person is not driven by internal forces, nor is he a passive reactor to external pressure. Rather, a person is both the agent and the object of environmental influence. Behavior is a function of

interdependent factors. Thus cognitions do not operate independently. In a complete analysis of the cognitive control of behavior, mediating processes *must* be tied to observable action. Furthermore, the determinants of these cognitive mediating processes *must* be spelled out. An instructive instance of this emphasis on reciprocal determinism of behavior is found in Bandura's (1977) concept of self-efficacy, an important mini-theory within the larger social learning framework. Originally proposed to provide a unifying account of the mechanisms responsible for the well-documented efficacy of different behavioral treatment methods for phobic disorders, self-efficacy theory states

FIGURE 1

Component processes in the self-regulation of behavior



Reprinted from A. Bandura, *Social learning theory*. Englewood Cliffs, NJ: Prentice-Hall, 1977. With permission.

that psychological procedures change behavior to the extent that they alter expectations of personal efficacy. These expectations reflect the subjective estimate that one has the ability to cope successfully with a threatening situation. They are differentiated from outcome expectations, which are defined as the client's belief that a particular behavior will result in a certain outcome.

Expectations of self-efficacy generalize to other situations and help explain the process of generalized behavior change. Another important prediction from this theory is that the strength of efficacy expectations will help determine how long clients will engage in active coping behavior in the face of obstacles and adverse psychological experiences. As such, this prediction bears importantly on the maintenance of treatment-produced improvement. Unless treatment creates strong expectations of efficacy, coping behaviors may be easily extinguished following the termination of therapy. The phenomenon of relapse is a problem for all methods of psychological treatment, including behavior therapy. Self-efficacy theory is a means of conceptualizing the relapse process and suggests procedures for facilitating the long-term maintenance of behavior change, especially in the addictive disorders (Marlatt & Gordon, 1985).

Efficacy expectations are based on four major sources of information: behavioral performance, vicarious experience, physiological arousal, and verbal persuasion. The theory relies upon cognitive mediating processes in conceptualizing behavior change mechanisms. Yet the evidence on the outcome of psychological therapies (Rachman & Wilson, 1980) provides strong support for the *a priori* prediction of social learning theory that performance-based treatment methods are most effective in altering efficacy expectations and thereby producing changes in behavior and emotional reactions.

2. HISTORICAL DEVELOPMENT OF THE THEORY

The initial statement of social learning theory as a systematic approach to behavior change was contained in Bandura and Walter's (1963) volume entitled *Social Learning and Personality Development*. However, it was the publication of Bandura's (1969) influential text *Principles of Behavior Modification* that marked the first major exposition of social learning theory as it has come to be widely known today. The theory grew out of the principles and procedures of classical and operant conditioning that by the 1960s had been demonstrated to be effective in the conceptualization and treatment of diverse clinical disorders (Eysenck, 1964; Ullmann & Krasner, 1965). Aside from drawing on these principles of learning, social learning theory incorporated most of the methodological emphases that the study and application of conditioning principles brought to clinical research and practice. The detailed specification of therapeutic techniques, the focus on behavior per se in assessment, treatment, and evaluation of therapy outcome, and the advance in measurement and methodology were all directly associated with the methodological behaviorism that characterized the conditioning approach.

Bandura (1969) argued that the early applications of learning theory to the treatment of clinical disorders were unnecessarily narrow because of the

reliance upon a limited range of learning principles that were derived from studies of animal conditioning. To account more adequately for the way in which more complex social behavior is acquired and modified, he found it necessary to introduce new principles derived from developmental, social, and cognitive psychology. The major innovations in Bandura's (1969) account of the principles of behavior modification were the emphases on symbolic events, vicarious learning, and self-regulatory processes, as indicated in the previous section.

During the 1970s, psychology as a whole grew increasingly cognitive in character. Since social learning theory was rooted in the content and methods of experimental psychology, it was both inevitable and desirable that it too became more cognitive in nature (Bower, 1978; Wilson, 1982a). The emergence of self-efficacy theory in 1977 was a clear example of this trend (see also Bandura, 1982a). During this decade social learning theory became the dominant conceptual approach within the rapidly expanding field of behavior therapy (Goldfried & Davison, 1976; Wilson & O'Leary, 1980).

What was true for therapeutic applications held also for advances in clinical assessment. Cognitive psychology played a key role in the expansion and refinement of the social learning analysis of personality development and assessment (Mischel, 1973). No one has had a greater conceptual impact on personality assessment in behavior therapy than Mischel, whose landmark 1968 book was erroneously greeted by both friend and foe as a situationist's manifesto of sorts that was designed to rid us of the notion of personality. Five years later Mischel refined his message and specified a series of "person variables" that were the product of social experience and cognitive growth. They included the individual's competencies to generate different behaviors under appropriate conditions; the person's encoding and categorization of events and people, including the self; his or her expectancies; the subjective values of any expected outcomes; and the individual's self-regulatory systems and plans.

2.1 SOCIAL LEARNING THEORY AND ALCOHOLISM: INITIAL FORMULATION

From the beginning, social learning theory had as its aims the conceptualization and modification of a wide range of normal and abnormal behavior (Bandura, 1969). Over time, the theory has been applied to an increasingly wide range of psychological phenomena, to educational problems, and even to medical conditions (Bandura, 1982a; O'Leary & Wilson, 1987). The analysis of aggression (Bandura, 1973) and the extension of the theory to the understanding and modification of fear and phobic reactions (Bandura, 1977) were particularly important in the development of the theory and demonstrations of its utility. The social learning analysis of the latter phenomena gave rise to the theory of self-efficacy, which, as discussed in a later section in this chapter, has been successfully extended to a variety of other phenomena, including the treatment of alcohol abuse (Bandura, 1982a; Marlatt & Gordon, 1985).

The landmark analysis of alcoholism in terms of social learning theory was presented in Chapter 8 of Bandura's (1969) text. Criticizing the then

prevalent psychological approaches to alcoholism that tried in vain to identify specific personality traits or psychodynamic conflicts that reliably differentiated alcoholics from non-problem drinkers, Bandura made the following observation:

Just as persons who differ markedly in personality attributes can learn to use tobacco excessively, so, given appropriate social-learning conditions, persons who possess diverse personality characteristics can be taught heavy drinking of alcoholic beverages. In fact, it has been shown repeatedly that no matter what deviant behavior is singled out for study, it is usually found in a wide variety of personality types. A much more fruitful approach to the understanding of alcoholism would be to investigate the learning contingencies specifically associated with drinking behavior and the reinforcement mechanisms maintaining self-intoxication. (1969, p.529)

He went on to discuss the effects of alcohol on emotional arousal and avoidance behavior (the tension reduction theory of alcohol use and abuse), and other determinants of alcohol consumption, with particular emphasis on the specific cultural and social conditions that might initiate and sustain different patterns of drinking. This analysis led to the following conclusion:

From a social-learning point of view, alcoholics are people who have acquired, through differential reinforcement and modeling experiences, alcohol consumption as a widely generalized dominant response to aversive stimulation. Therapeutic attention would therefore be most profitably directed toward reducing the level of aversive stimulation experienced by individuals, and toward eliminating alcohol stress responses either directly or, preferably, by establishing alternative modes of coping behavior. Given more effective and rewarding means of dealing with environmental demands, individuals will have less need to resort to self-anesthetization against everyday experiences. (Bandura, 1969, p.536)

Presciently, Bandura observed that the stress reduction theory of alcoholism that was the first behavioral model of drinking had possibly been given too much explanatory weight and that other psychological determinants of alcohol abuse remained to be identified. The following decade of research confirmed this prediction in demonstrating several different psychosocial determinants of drinking (Marlatt, 1978; Nathan & Briddell, 1977).

In summarizing what was known about the application of learning principles to treating alcoholism, Bandura (1969) had little more to draw on than the results of various aversive conditioning methods (e.g., Lemere & Voegtlin, 1950). In the late 1950s and the 1960s, behavior therapy for antisocial disorders or problems of lack of self-control, such as alcoholism, was restricted almost exclusively to aversion conditioning methods. Social learning theory was instrumental in broadening this narrow focus and moving away from a reliance on aversion therapy (Wilson & Davison, 1974). Thus Bandura (1969) emphasized the need to supplement these methods with multiform treatment comprising the development of vocational and interpersonal competencies through modelling and reinforcement procedures, the modification of dysfunctional self-reinforcement patterns, desensitization of stress-provoking situations, and even a social-systems approach to the problems of widespread alcoholism in particular subcultures. At that time there was virtually no

research along these lines, but the prescription proved to be a blueprint for behavioral researchers in succeeding years, as the remainder of this chapter indicates.

The social learning analysis of alcohol use and abuse in this chapter draws not only on research inspired directly by Bandura's (1969, 1977) theorizing, but also on closely related learning-based or behavioral studies, even though they might not have been explicitly identified as "social learning theory." A landmark in the treatment of alcoholism along social learning lines was Lazarus's (1965) innovative case report of the clinical practice of "broad-spectrum" behavior therapy with an alcoholic. In this approach Lazarus combined systematic desensitization with a variety of other behavioral techniques designed to modify both the alcoholic's excessive drinking and behavioral problems associated with the drinking. Among the components of Lazarus's broad-spectrum behavior therapy for alcoholism were medical care to treat alcohol-related physical disabilities; aversion conditioning to modify the patient's drinking behavior directly; tests and interviews to identify specific stimulus antecedents of anxiety (to permit construction of anxiety hierarchies for systematic desensitization); assertion training; behavioral rehearsal; hypnosis to counter-condition anxiety-response habits; and involving the patient's wife, to help her see and alter her role in the patient's alcoholism. Lazarus (1971) not only pioneered the clinical expansion and development of behavior therapy in general, but also anticipated the now routine use of multifaceted treatment programs with problem drinkers.

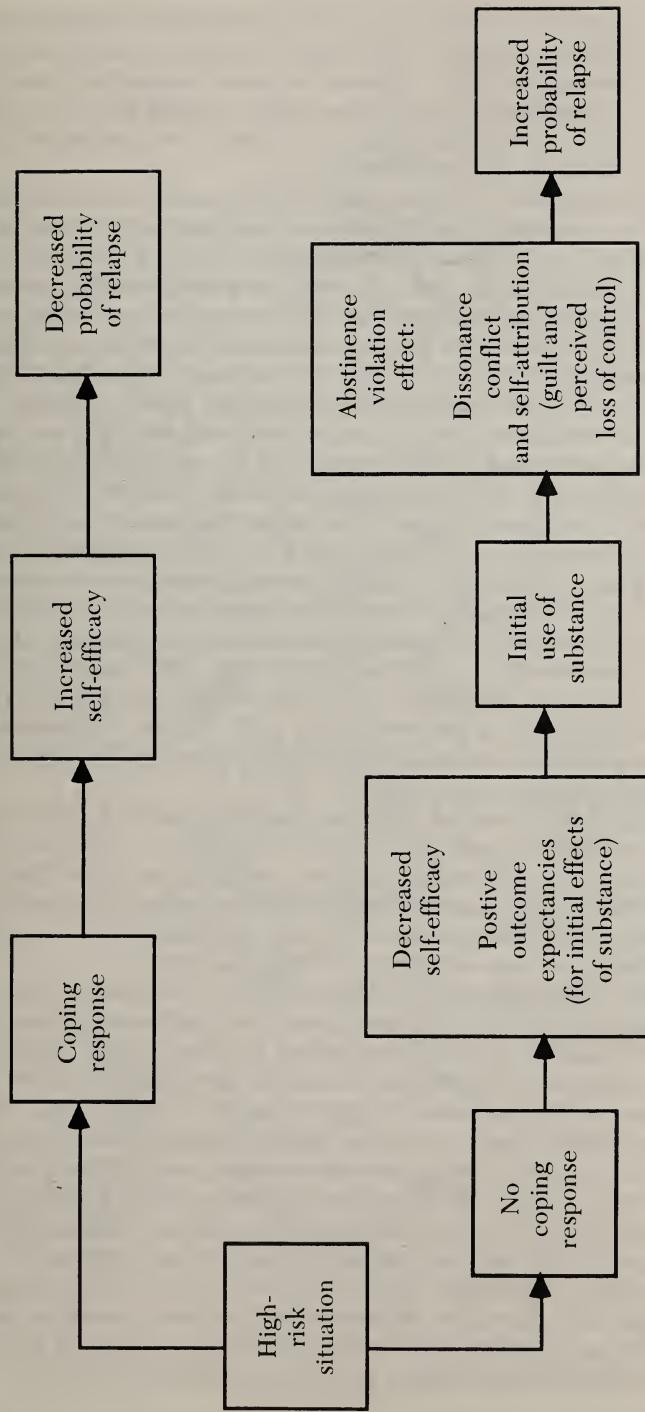
Although other conceptions of behavior therapy do not incorporate all elements of social learning theory, particularly its more cognitive constructs (Eysenck, 1982; Wolpe, 1976), social learning theory has provided a conceptual framework for much of the clinical expansion of behavior therapy (Goldfried & Davison, 1976; Lazarus, 1971; O'Leary & Wilson, 1987). Furthermore, it can be argued that the recent history of behavior therapy is the history of the application of social learning theory to clinical problems.

2.2 EXPANSION AND REFINEMENT OF SOCIAL LEARNING THEORY OF ALCOHOLISM IN THE 1970s

In his 1969 book, Bandura emphasized that the evaluation of psychological treatments should distinguish among the initial induction of behavioral changes, their generalization to the natural environment, and their maintenance over time. Different variables may govern each of these processes, and generalization and maintenance can be ensured only to the degree that procedures explicitly designed to accomplish such objectives are built into the overall treatment program. The focus on maintenance of behavior change, which had typically been ignored in psychological treatment approaches, heralded a significant conceptual development. One of the major advances in the social learning account of alcoholism in the 1970s was Marlatt's relapse prevention model, a detailed analysis of the psychological processes governing maintenance of behavior change in alcoholics (Marlatt, 1978; Marlatt & Gordon, 1985). Marlatt's model is shown in Figure 2.

FIGURE 2

Cognitive-behavioral model of the relapse process



Reprinted from G.A. Marlatt and J. Gordon (Eds.), *Relapse prevention*. New York: Guilford, 1985.

Even after successful treatment, the abstinent alcoholic is faced with specific high-risk situations. The model, in essence, states that the lack of a response for coping with a high-risk situation initiates a chain of events in which a decreased sense of self-efficacy leads to initial use of the alcohol. Initial consumption is even more likely to occur if the individual has positive expectations for the alcohol effect.

The abstinence violation effect (AVE) occurs when the individual is personally committed to an extended or indefinite period of abstinence and a slip, or lapse, occurs during this time period. The AVE is a cognitive-affective reaction to an initial slip that influences the probability that the lapse will be followed by increased substance use. There are two components to the AVE: a cognitive attribution as to the perceived cause of the lapse coupled with an affective reaction to this attribution. An increased AVE is postulated to occur when the individual attributes the cause of the lapse to internal, stable, and global factors that are perceived to be uncontrollable (e.g., lack of willpower and/or the emergence of the symptoms of an underlying addictive disease). The intensity of the AVE is decreased, however, when the individual attributes the cause of the lapse to external, unstable (changeable), and specific factors that are perceived to be controllable (e.g., a transitory deficit in coping with a specific high-risk situation). Negative (internal, stable) attributions undermine self-efficacy and increase outcome expectations of continued failure, a usually fatal combination of factors leading to resumption of substance abuse.

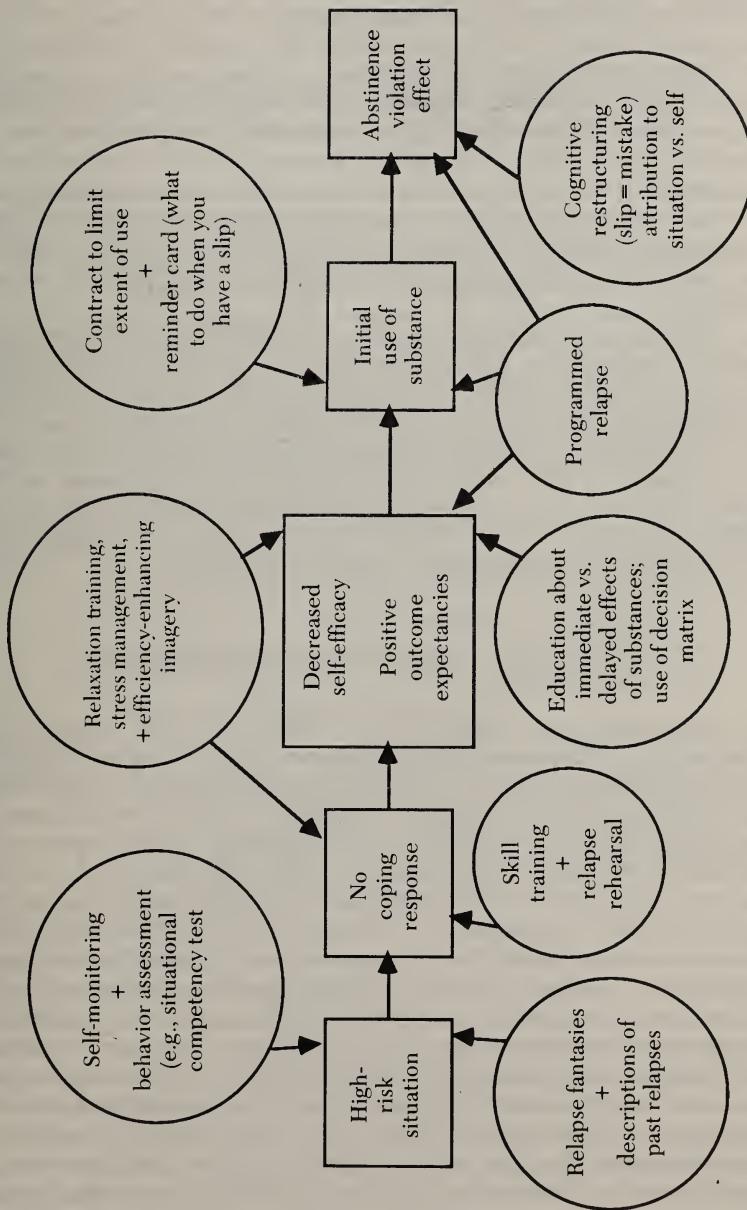
In characteristic social learning fashion, this analysis of the relapse process has direct implications for intervention, namely, the cognitive or behavioral procedures for avoiding an AVE. Figure 3 describes a range of cognitive and behavioral methods for negating the AVE at different points in the assumed sequence of events.

Cognitive behavioral treatments are designed to develop coping skills, restore self-efficacy, and create more constructive attributions. The latter would emphasize external (situational), unstable, and controllable causes of slips or failures. The usual strategy in cognitive behavior therapy is to encourage clients to attribute setbacks to external, unstable, and usually specific determinants, and successful coping to internal factors.

As indicated above, social learning theory has been closely associated with important advances in the analysis and treatment of fear and phobic disorders (Bandura, 1977). Over the past 25 years, reliably effective methods for the treatment of phobic and obsessive-compulsive disorders have been developed within behavior therapy (Barlow & Wolfe, 1981; Rachman & Hodgson, 1980). These techniques, such as systematic desensitization, flooding, *in vivo* exposure, and response prevention, were all derived from animal conditioning research, although the most recent social learning account is in terms of self-efficacy theory (Bandura, 1982a). In an extrapolation from a social learning analysis of fear and phobic avoidance to alcoholism, Wilson (1978b) suggested that self-efficacy theory was well suited to analysing processes of maintenance of therapeutically produced behavior change in the addictive disorders, and Marlatt and Gordon (1985) have assigned self-efficacy an important place in their relapse prevention model (see Figure 3).

FIGURE 3

Relapse prevention: specific intervention strategies



Hodgson and his colleagues, drawing on notions of classical conditioning and avoidance learning, have also drawn a significant parallel between the behavioral treatment of phobic and obsessive-compulsive disorders and the analysis and treatment of alcoholism (Hodgson et al., 1979; Stockwell et al., 1982). Hodgson et al. (1979) suggest that the two seminal concepts in the alcoholism field of craving and loss of control translate readily into "fear" and "avoidance behavior" in the assessment and treatment of phobic and obsessive-compulsive disorders. They define craving as a complex system of interrelated responses involving subjective, physiological, behavioral, and biochemical components that are sometimes only partially coupled (Rachman, 1978). Viewed in this fashion, craving is a learned set of responses that can be extinguished in the same manner as fear reactions are extinguished in phobic patients. More specifically, recommended treatment methods would be based on systematic, prolonged exposure to the cues that elicit craving, together with the prevention of the consumption of alcohol that typically accompanies craving.

There is a way in which social learning theory appears to provide a more complete explanation of these data than one based on conditioning factors alone. Stockwell et al. (1982) showed that alcoholics who are not that physically dependent are responsive to cognitive influences, whereas those who are severely dependent are relatively unresponsive to such influences. Here they are proposing a dual system of control over craving, much as Bandura (1969) suggested that negative emotional reactions may be the function of two different systems — one of which is self-generated (i.e., cognitive) and the other evoked directly by conditioned aversive stimuli.

3. CHARACTERISTIC RESEARCH METHODS

As noted above, social learning theory is rooted in methodological behaviorism, with its commitment to operational specification of procedures, objective behavioral measurement, and the experimental method. The particular experimental methodologies employed by social learning theory run the gamut from various types of between-group designs to single-case experimental designs (Bandura, 1969; Kazdin, 1980). The flexible use of different research strategies in the development and application of social learning theory has been necessitated by its broad scope and the range of levels of research it encompasses. This latter point about different levels of research is particularly important. As an overall, integrative theory of normal and abnormal behavior, social learning theory attempts not only to explain the basic psychological mechanisms involved in behavior change but also to generate and guide the use of methods that effectively alter dysfunctional or maladaptive behavior. As such it is nourished by both basic and applied research. The assumption is that basic psychological research can significantly influence the development of effective treatment methods, and the clinical practice, in turn, should help shape the nature and directions of basic research. To realize the benefits of this reciprocal influence — that is, to close the endlessly discussed

gap between research and clinical practice — it is imperative that we develop an integrative model or framework that spells out the respective contributions of basic and applied research to the overall objective of improving our understanding, treatment, and prevention of alcohol abuse (Wilson, 1985).

TABLE 1

Different levels of scientific/clinical analysis along the continuum from basic research to clinical practice

<i>Level 1</i>	Basic laboratory research on mechanisms of behavior change (e.g., studies using the balanced placebo design to separate the cognitive and pharmacological effects of drinking, such as Marlatt et al.'s [1973] investigation).
<i>Level 2</i>	Analogue treatment research to identify effective ingredients of therapeutic procedures under controlled laboratory conditions (e.g., studies of aversion therapy using the taste test as a dependent measure, such as Wilson et al.'s [1975] investigation).
<i>Level 3</i>	Controlled clinical research with patient populations. Two subtypes can be distinguished: (a) studies of specific techniques with detailed process and outcome measures (e.g., Chaney et al., 1978), and (b) studies designed to show optimal treatment effects using multicomponent therapy packages (e.g., Azrin, 1977).
<i>Level 4</i>	Clinical practice. Therapists may measure outcome in case studies or clinical series (an AB design), or, in rare instances, conduct single-case experimental designs (e.g., Lemere & Voegtlin's [1950] evaluation of the effects of chemical aversion therapy with alcoholics).

Table 1 outlines a framework that incorporates the different levels of analysis along the continuum from basic research to clinical practice in the development of effective treatment methods. This framework emphasizes that each of the four levels has a unique and necessary role in the development of an applied clinical science. Clearly, the flow of influence along this continuum is not unidirectional. There is a reciprocal interaction between different levels. For example, novel clinical observations or uncontrolled findings by practitioners at level 4 often suggest new treatment techniques and indicate the limitations of existing methods. These observations would influence the research done at levels 1 through 3.

3.1 LABORATORY RESEARCH

Laboratory research at level 1 on basic psychological mechanisms has witnessed major advances as a result of the activities of social learning and other behaviorally oriented investigators. Offfundamental importance was the development of behavioral measures of actual drinking by alcoholics and nonalcoholics in controlled laboratory settings (Mendelson & Mello, 1966; Nathan & O'Brien, 1971). Succinctly summarized by Pomerleau et al. (1976), this research

employed an operant-conditioning paradigm in which a quantifiable behavior, such as bar-pressing, produced a predetermined amount of alcohol as a consequence. In specially designed research wards, the drinking behavior of volunteer alcoholics (screened to exclude those for whom further drinking was medically contraindicated) was studied objectively over an extended period of time. Predrinking, drinking, and postdrinking intervals were designated, with some studies lasting up to several weeks. Observations of social behavior as well as psychiatrically relevant behavior were typically conducted using one-way mirrors and closed-circuit television. Blood alcohol determinations were made at regular intervals together with various metabolic tests. (p.852)

Research using this innovative albeit controversial methodology constituted a major breakthrough in the investigation of alcoholism. Prior to these behavioral studies, research on alcoholics' drinking was confined primarily to retrospective self-reports about drinking, with all the problems inherent in such a subjective approach, or to investigations of alcoholics only when they were sober. For the first time, direct behavioral observations and analyses of actual drinking by alcoholics were made, permitting rigorous examination of seminal assumptions about the nature and treatment of alcoholism that had uncritically passed into the lore. This behavioral methodology enriched our understanding of the causes, correlates, and consequences of excessive drinking, forcing major revisions in such key concepts as craving and loss of control, and challenging the theoretical and empirical bases of the disease theory of alcoholism proposed by Jellinek (1960).

Behaviorists have tended to reject the concept of craving as a superfluous construct because it is inferred from the very behavior, namely loss of control over drinking, that it has been used to explain within the disease theory of alcoholism (Mello, 1975). More recently, however, as noted earlier in this chapter, Hodgson and his colleagues have conceptualized craving in learning terms and initiated a fruitful program of research on its psychobiological determinants and its effects on subsequent alcohol consumption (Hodgson et al., 1979; Stockwell et al., 1982). Fundamental to this productive program of research was the development of an operational measure of craving that was suitable for both observational and experimental studies. They have shown that speed of drinking, especially the first drink, is a useful and sensitive measure of craving that is sensitive to differences in prior duration of abstinence as well as degree of physical dependence on alcohol in alcoholics.

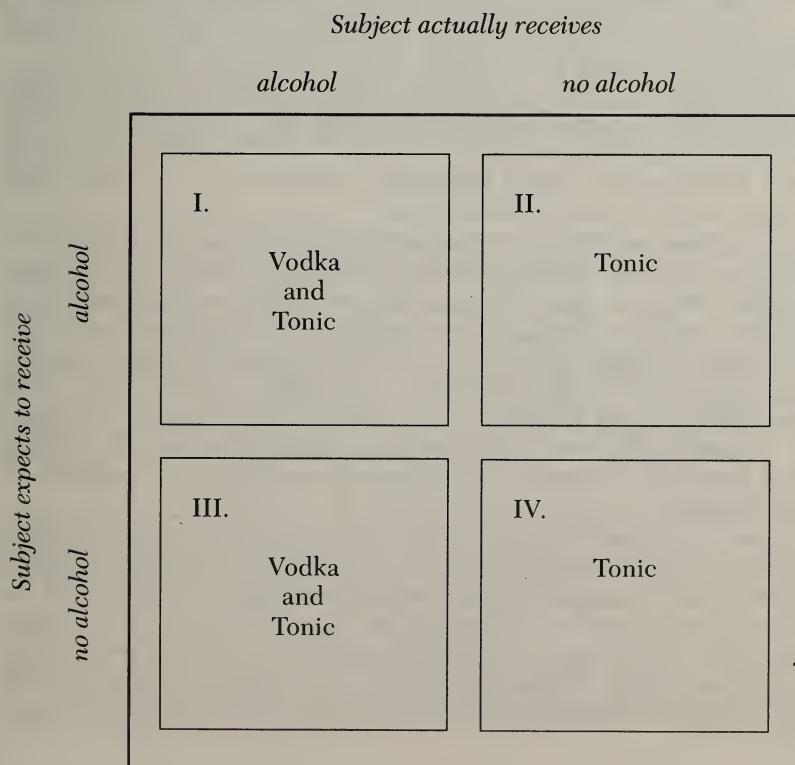
A second behavioral strategy for assessing alcohol consumption in the laboratory that has proved central in the development of a social learning analysis of drinking is the "taste-rating task" (Marlatt, 1976). This taste test, derived from a similar procedure used by Schacter et al. (1968) to measure food

intake, was introduced into the study of alcohol use and abuse independently by Marlatt (Marlatt et al., 1973) and P.M. Miller (1976; Miller & Hersen, 1972). Briefly, taste tests require subjects to taste a variety of available alcoholic (and possibly nonalcoholic) beverages and to rate them on a variety of taste dimensions. Subjects are instructed to sample as much of the beverages as they wish in order to make judgments that are as accurate as possible. The test is really a surreptitious measure of how much alcohol is consumed in a limited time period.

The taste-rating task has been incorporated into another prominent research method that has characterized the social learning analysis of the causes and consequences of alcohol consumption — the balanced placebo design (Marlatt et al., 1973; Marlatt & Rohsenow, 1980). Initially described but neglected in the 1960s (Lyerly et al., 1964), this experimental design was rediscovered a decade later by Marlatt. His studies and theorizing helped to initiate a series of research programs that have advanced the experimental analysis of the influence of cognitive set on drinking and raised serious questions about traditional conceptions of alcohol use and abuse.

FIGURE 4

The Balanced Placebo Design



The logic of the balanced placebo design is obvious. In the past, studies of alcohol (indeed, all types of drugs) consisted essentially of comparisons between Groups I and II as shown in Figure 4. The actual drug was compared to a chemically inert placebo substance. Nevertheless, both groups expected that they had received the drug itself. Expectations were presumably equated but not systematically manipulated. Groups III and IV are necessary in order to study the independent effects of expectations about drug ingestion. This design has proved invaluable in studying the effects of anticipated consequences (outcome expectations) of drinking, the findings on which are discussed in §5.1.1. Of course, there are limitations to this experimental design. It has been used successfully at relatively low levels of blood alcohol concentration (up to 60 mg% or thereabouts), but there is some question about its utility at higher levels of blood alcohol concentration (e.g., Levenson et al., 1980).

Finally, although a social learning analysis of alcohol use and abuse relies primarily on studies conducted on non-problem and problem drinkers, laboratory research with animals is important to the extent that it elucidates the role of learning in the acquisition and maintenance of alcohol consumption. An instructive example of such research, discussed below, is that of Siegel and his associates on classical conditioning and the development of tolerance in rodents (Poulos et al., 1981).

3.2 ANALOGUE TREATMENT RESEARCH

The methodological and practical difficulties of evaluating the efficacy of psychological treatments by studying therapy carried out by experienced practitioners with actual clients under clinical conditions are well known (Rachman & Wilson, 1980). It is hard and often impossible to conduct highly controlled research in service-delivery settings, and it is difficult to recruit experienced therapists who have the time or desire to participate in such research. Furthermore, finding sufficiently large numbers of similar clients with common problems is a formidable task.

Laboratory-based study, or analogue research as it has come to be known, provides an alternative research strategy to the direct study of clinical situations. The strategy is to evaluate specific treatment methods applied to well-defined, circumscribed problems under controlled laboratory conditions. The methodological advantages and disadvantages of analogue research have been discussed in detail elsewhere (e.g., Borkovec & Rachman, 1979; Kazdin, 1980). The fact that different parameters of the treatment technique can be systematically varied and different components selectively analysed makes it possible to determine the necessary and sufficient conditions of treatment success.

Behavioral research on the effects of aversion therapy with alcoholics provides an example of analogue research. Since the use of aversion therapy is predicated on the assumption that it suppresses drinking by endowing the alcohol with conditioned aversive properties, it was necessary to demonstrate such aversions in a laboratory setting before testing their efficacy in a complex clinical outcome study. This initial laboratory-centred approach permitted the rigorous, objective measurement necessary to specify the processes responsible

for behavioral change, without the complicating influence of the many other factors inherent in clinical treatment programs that feature aversion therapy. Accordingly, Wilson et al. (1975) assessed the effects of the electrical escape conditioning procedure on free-operant drinking baselines in a semi-naturalistic situation that allowed prolonged drinking. Comparison of an aversion conditioning procedure with a control procedure revealed that neither method had any significant effect on alcohol consumption. P.M. Miller et al. (1973) completed a similar study with similar results, using the taste test as a behavioral measure of electrical aversion conditioning's effects. Given that this technique failed to modify drinking even in the laboratory setting, one largely free of the various sociopsychological pressures associated with drinking in the real world, it could be concluded that this form of aversion conditioning was ineffective.

Within the context of the controlled laboratory setting, Cohen et al. (1971) demonstrated that chronic alcoholics could, within a free operant situation, voluntarily restrict their drinking below five ounces per day if this moderation was positively reinforced by access to an enriched environment as opposed to remaining in an impoverished environment. These studies demonstrated a functional relationship between drinking and its environmental consequences. Treatment programs making use of this principle are necessarily more complex and multifaceted than the simple procedures employed in the laboratory. The efficacy of these programs must be assessed directly in research at a different level of analysis — level 3 in Table 1.

To summarize, once behavior change principles have been demonstrated in analogue research, they can be extended to and evaluated in less controlled clinical settings, where the questions of comparative efficacy and cost effectiveness among different treatment methods are of paramount importance.

3.3 CLINICAL RESEARCH

A major contribution of behavior therapy has been the adoption and refinement of several innovative research strategies for the analysis and evaluation of treatment outcome. These research strategies range from single-case experimental designs to a variety of different group designs for the evaluation of complex therapeutic programs in clinical settings (Kazdin, 1980; Kazdin & Wilson, 1978). These different research strategies go far beyond traditional research methods in clinical psychology and allow more refined empirical analyses of specific treatment techniques under different conditions.

Single-case experimental designs, which have featured prominently in research at levels 1 and 2, have been seldom used in clinical outcome research. This neglect is surprising in view of their extensive application in treatment research with other complex clinical disorders (Hersen & Barlow, 1976).

The multi-component treatment program research strategy is a problem-oriented research strategy that allows for an evaluation of multiple treatment methods that are typically used in clinical practice. The primary emphasis is on producing clinically significant change, and thus it is close to the nature and purpose of clinical practice. It is logically one of the first means to

evaluate a given treatment approach, and is exemplified in the pioneering research of Sobell and Sobell (1973, 1978) and Azrin (1976), which is discussed below (§5.2.4). If the multi-component treatment program proves to be successful, subsequent research can be directed towards identifying the effective components of the overall package. If it fails to produce therapeutic improvement, it makes little sense to try to analyse its component parts. In other words, it serves as a useful screening procedure in determining whether further component analysis research is warranted.

In the dismantling research strategy, specific components of the treatment package are systematically eliminated, and the associated decrement in treatment effects is measured. The relative contributions of each component to the total treatment program can then be assessed (e.g., W.R. Miller et al., 1980). Both the separate and combined effects of components can be analysed. It can also be determined whether these effects are interactive or additive.

Lastly, there is a technique-focused strategy that investigates a particular treatment method against specific control groups with detailed process and outcome measures, as in the evaluation of the effects of social skills training by Chaney et al. (1978).

The specific methodological contributions of the social learning or behavioral approach to the treatment of alcoholism have been extensively documented by Sobell and Sobell (1983). These include the quantification of daily drinking behavior, frequent follow-up assessments, research on the validity of alcoholics' self-report of drinking, and measures of drinking for use in epidemiological studies of alcohol's effects. Two of these methodological contributions must be underscored. The first is the rejection of the simplistic dichotomous outcome measure of "dry or drinking" that had characterized much of traditional alcoholism treatment research. It is now clear that evaluation of treatment programs in terms of whether alcoholics are abstinent ("dry") or not ("drunk") is too restrictive and based on questionable assumptions about the nature of alcoholism (Nathan & Briddell, 1977; Pattison, 1976). Aside from abstinence, modified or controlled drinking may be a realistic goal of treatment in some cases of less severe dependence on alcohol. The quantification of drinking evaluates the level rather than the mere occurrence of consumption and therefore provides a more sensitive index of treatment effects. Just as the "dry or drinking" outcome dichotomy derived logically from the traditional disease conception of alcoholism, so the use of a continuous rather than an "either/or" measure of drinking by behavioral researchers was demanded by the social learning model of alcoholism with its emphasis on drinking behavior, the conditions under which it occurs, and any changes over time. Sobell and Sobell (1983) point out that prior to 1970 no major empirical study had measured the actual amount of daily drinking by alcohol abusers. Significantly, the first study to measure daily drinking behavior (Sobell & Sobell, 1973, 1978) did so because of a need to assess the achievement of non-problem drinking.

Second, the use of multiple measures of outcome has significantly advanced the field. Since abstinence does not ensure rehabilitation of the alcoholic (Pattison, 1976), multidimensional measurement of outcome is

desirable, and may focus on physical health, emotional, social, and vocational adjustment in addition to the primary data of the amount and pattern of alcohol consumption. An impressive feature of the Azrin (1976), Hunt and Azrin (1973), and Sobell and Sobell (1978) studies is the extent to which they measured these variables.

4. BOUNDARIES OF THE THEORY

Social learning theory encompasses a broad range of alcohol-related phenomena. First, it bids fair to explain all alcohol consumption. All drinking is conceptualized along a continuum from normal to abnormal and is explained by a common pool of psychological principles. Evidence for this is summarized in the next section. Second, the theory accounts for both individual and group drinking patterns. At the most general level, social learning variables are reflected in the cultural norms that define the learning contingencies governing alcohol consumption; the influence of cultural mores on drinking patterns is widely acknowledged. Varying patterns of modelling and reinforcement provided by key socializing agents, particularly the family, can account for the fact that only a minority of drinkers become alcoholic, and, furthermore, could in principle account for the fact that alcoholism runs in families. Finally, social learning theory addresses itself to the development and maintenance of drinking, and is hence relevant to both modification and prevention of drinking problems.

What then does it omit? Characteristically, social learning theory places little emphasis on genetic factors. The continuum view of drinking clearly demarcates this theory from genetic theorizing such as Goodwin's (1976). His position is that only alcoholism is hereditary, whereas heavy social drinking and even problem drinking (as distinguished from true "alcoholism") are determined by environmental factors. In this sense, genetic theory and social learning theory are fundamentally incompatible in accounting for alcoholism.

5. RESEARCH RESULTS AND NEEDS

5.1 SOCIAL LEARNING DETERMINANTS OF DRINKING AND ALCOHOL'S EFFECTS

Research inspired by social learning theory has significantly expanded our knowledge about the development, maintenance, and modification of alcohol use and abuse. Research on the determinants of alcohol consumption has established that drinking is heavily influenced by different psychosocial variables. These include the following: antecedent environmental cues, which may, through classical conditioning, elicit the urge to drink; the behavioral consequences of drinking, which may variously act as positively reinforcing, negatively reinforcing, or punishing stimuli; vicarious learning, in which the person models the drinking behavior of significant others; person variables,

such as social skills or competency in coping with interpersonal conflict; self-regulatory processes; and cognitive factors such as learned expectations.

The effects of alcohol consumption, it has been shown, can vary greatly as a complex function of diverse psychosocial influences. These include the person's social learning history; his/her cognitive set, such as expectations or beliefs about alcohol's effects; and the physical and social setting in which drinking occurs.

In what follows, illustrative research methods and results are summarized from three major areas of experimental inquiry: namely, the relationship between alcohol and stress, alcohol's effects on sexual responsiveness, and social modelling and drinking. These three areas not only encompass significant experimental research but also address important methodological and conceptual issues.

5.1.1 *Alcohol's Effects on Tension or Stress*

The most remarkable feature of the tension reduction theory of alcohol use and abuse is how widely it is believed. The notion that alcohol reduces tension and that people drink in order to obtain this effect appears to make common sense and is deeply ingrained in folklore and clinical experience. Brown et al. (1980) conducted a factor analysis of the expectancies associated with moderate alcohol consumption in a diverse group of non-problem drinkers and found that six factors emerged, one of which corresponded to tension reduction. Brown (1985) found that a measure of college students' outcome expectations about the tension-reducing effects (both physical and mental) of alcohol was the strongest predictor of problem drinking in this population. She also found that alcoholics reported more tension-reduction effects of alcohol than other drinkers. Tension reduction was given as a reason for drinking by a representative sample of men living in a London suburb in a study by Edwards (1972). Problem drinkers in this sample tended to show high scores on this tension-reduction factor. Deardorff et al. (1975) similarly found that this factor was one of three factors that distinguished between the problem and non-problem drinkers. These findings, showing that outcome expectations of tension-reducing effects of alcohol are associated with or predict problem drinking, are consistent with social learning theory's emphasis on anticipated consequences as a determinant of behavior. Clinicians also share the view that alcohol reduces tension (Cappell, 1975). Laboratory studies on the effects of alcohol on stress, however, have revealed a more complex set of findings than the commonsense view of tension reduction.

The tension reduction theory of drinking appears to be based on two assumptions: (1) that alcohol consumption reduces tension, and (2) that this tension-reducing effect motivates drinking. With respect to the first assumption, any overview of the laboratory evidence on this topic must conclude that no consistent pattern of alcohol's effects on tension has been demonstrated (Cappell, 1975). Alcohol has been variously shown to increase, decrease, or not affect tension in human subjects (Wilson, 1982b). These apparently conflicting data are surprising only if it is assumed that there is an automatic, invariant relationship between alcohol and stress reduction.

According to a social learning analysis, behavior commonly attributed to alcohol is related to it in a complex fashion. Among the many variables that determine the effect of alcohol on tension and other emotional states are the amount of alcohol that is consumed, the person's prior experience with alcohol, individual differences based on physiological responsiveness to ethanol and specific social learning histories, learned expectations about alcohol and its effects, and the social setting in which drinking occurs (Marlatt, 1987; Sher & Levenson, 1983; Wilson, 1982b). In view of the influence of all these psychosocial and pharmacological factors on the relationship between alcohol and stress (as well as other social behavior and emotional responses), it is simply no longer useful to ask the question: Does alcohol reduce tension? The more meaningful question to pose is: Under what conditions, at which doses, in whom, on what measures, and how does alcohol affect specific forms of tension? (Wilson, 1982b).

Use of the balanced placebo design to study outcome expectations has yielded a consistent finding of gender differences in the anticipated tension-reducing effects of alcohol (Abrams & Wilson, 1979; Marlatt, 1987). Men expect that alcohol will decrease their social anxiety in heterosexual situations and enhance sexual arousal (Wilson, 1981). Consistent with this expectation, men are likely to approach women more readily when intoxicated. It follows from this social learning analysis that men who know that their female companion has been drinking will feel less social anxiety and view her as more open to sexual advances. Wilson et al. (1981) have confirmed the first of these predictions. These data provide an explanation for the observed gender differences in anticipated and experienced social anxiety (and sexual arousal; Wilson & Lawson, 1978). Women, as a result of a social learning history in which men often act unpredictably and aggressively when intoxicated, and when their own culturally acquired outcome expectations suggest that alcohol will disinhibit their self-control, feel threatened and come to be more defensive in heterosexual situations featuring drinking.

The second assumption of the tension-reduction theory is that this effect of alcohol motivates drinking. As noted earlier, both social and problem drinkers assert that this is one of the major reasons for drinking. The laboratory evidence on this part of the tension reduction theory is not free from conflicting findings, although some studies provide strong support for the notion. For example, Higgins and Marlatt (1975) showed that anticipation of interpersonal evaluation produced significantly increased consumption of alcohol. An earlier study by the same researchers, using threat of electric shock as the source of anxiety, failed to show significant results (Higgins & Marlatt, 1973). These data suggest that drinking will increase only in those situations perceived to be stressful and for which the individual expects alcohol to reduce the experience of tension or stress. This explanation fits well with social learning theory, with its emphasis on individual assessment and the role of situationally specific influences (Mischel, 1968).

Marlatt's (1978; Marlatt & Gordon, 1985) pioneering analyses of the psychosocial determinants of relapse in successfully treated alcoholics (discussed below) revealed that one of the major social stress experiences,

accounting for almost a third of the relapses studied, was the experience of frustration and anger, often arising in an interpersonal context. Instead of expressing these feelings of anger or dealing with them in a constructive manner, these patients began drinking again. In the laboratory Marlatt et al. (1975) demonstrated that both male and female social drinkers, when angered by a confederate in a work situation, drank significantly more alcohol in a taste-rating task than did control subjects who were not angered. This study also investigated the consequences of allowing angered subjects to "retaliate" by asserting themselves with the confederate. Subjects allowed to retaliate drank significantly less than subjects without the opportunity to retaliate. These results indicate that heavy drinkers who are provided with an alternative means of coping with a social stress frequently associated with drinking will show reduced alcohol consumption.

5.1.2 *Alcohol and Human Sexual Responsiveness*

In general, after drinking alcoholic beverages people tend to become more extraverted, express emotions more readily, show less social restraint, be more easily provoked into displaying aggression, and be less inhibited sexually. Alcohol has been linked to greater sexual activity and is usually associated with sexual offences such as rape and pedophilia. Despite the lack of direct evidence, the assumption is often that alcohol causes these antisocial actions. These changes in personal conduct are typically seen as support for the disinhibition theory of alcohol's effects. According to this popular view of intoxication, alcohol has a specific behavioral action.

The disinhibition theory of alcohol's actions on sexual behavior has taken one of two forms. The first hypothesizes a pharmacological action of alcohol in anesthetizing higher cortical brain mechanisms that ordinarily, in the sober state, control activities such as sexual behavior. An alternative expression of the theory relies on the psychodynamic notion of loss of superego functions of self-control over id forces. Thus the superego has been defined as that part of the psyche that is soluble in alcohol. Whether it is interpreted in specific physiological terms or in the psychodynamic sense of the superego solvent, the effect of alcohol reduces to its releasing otherwise restrained sexual behavior.

A social learning analysis emphasizes the roles of dose, personal history, and expectations in generating alcohol-related behavior. Overall, there is a significant negative linear relation between increasing BALs and physiological measures of sexual arousal in both men and women. At sufficiently high BALs (certainly 100 mg% or more), alcohol greatly impairs erectile capacity and ejaculation in men and vaginal blood pressure and orgasm in women (Wilson, 1981), and its chronic abuse impairs the biological bases of sexual function (Van Thiel & Lester, 1974). At lower BALs, however, laboratory studies of alcohol's effects on sexual responsiveness have demonstrated that beliefs about the consequences of intoxication can override the pharmacological properties of ethanol in determining behavior. Within a balanced placebo design, Wilson and Lawson (1976) demonstrated that male social drinkers who believed that they had consumed alcohol manifested significantly greater sexual arousal than those who believed that they had consumed only tonic water, while actual beverage content had no effect. This finding has been replicated and extended

by Briddell et al. (1978). Male social drinkers listened to tape-recorded descriptions of heterosexual intercourse, forcible rape, and non-sexual sadistic aggression. Subjects who believed that they had consumed alcohol experienced greater levels of penile tumescence than subjects who believed that they were sober; alcohol per se had no significant effect. In contrast to those who believed they were sober, subjects who believed that they had consumed alcohol were as aroused by the account of forcible rape as by the account of heterosexual intercourse.

Section 1 of this chapter stressed that the social learning account of self-control places a major emphasis on self-generated cognitive processes, such as self-evaluation. People usually strive to avoid personal actions that would cause them to evaluate themselves negatively. However, as noted before, self-evaluation is influenced by different psychosocial factors, which are often a specific function of the situation in which behavior occurs. For example, attributing socially disapproved conduct to alcohol's effects is an ideal means of evading or at least softening negative self-evaluation. McCaghy (1968) has suggested how alcohol allows child molesters to admit their deviant behavior yet exonerate themselves from personal responsibility. The time-honored excuse "If I had been sober it would never have happened" is a classic illustration of the resort to professed intoxication in the misattribution of culpability for transgressions from the accepted norms of conduct. This principle of alcohol-attribution versus self-attribution of responsibility can accommodate much of the available evidence on the effects of alcohol, or the effects of the belief that alcohol has been consumed, on various aspects of sexual behavior. It is directly applicable to the assumed association between drinking and sexual crimes such as rape or pedophilia, as McCaghy points out. Briddell et al.'s (1978) data on normal males' sexual response to rape-related sexual stimuli when they believed that they were intoxicated can be understood in the same way.

5.1.3 Social Modelling and Drinking Behavior

Anecdotal evidence has long suggested that some drinking habits are vicariously learned. In 1969 two influential theoretical contributions independently assigned an important role to modelling in determining drinking and subsequent behaviors: Bandura's social learning theory, and MacAndrew and Edgerton's anthropological theory. Nonetheless, the field had to wait until 1975 for the publication of the first experimental study of modelling effects on drinking (Caudill & Marlatt, 1975). Male social drinkers were asked to participate in a taste-rating task in which they made judgments about the taste characteristics of three wines. Ratings were made in the presence of a confederate of the experimenter whose rate of consumption was systematically varied. During the 15-minute taste-rating task, subjects were exposed to either a heavy consumption model (700 mL of wine), or a light consumption model (100 mL of wine), or no model. Subjects exposed to a heavy drinking model consumed significantly more than subjects exposed to a light drinking model or to no model. These results were replicated by Garlington and Dericco (1977).

Reid (1978) showed that the modelling effects occur not only in the artificial environment of the laboratory but also in a natural barroom setting. Subjects in Reid's study were male patrons of a cocktail lounge exposed to "warm" or "cold" male models who consumed alcohol at a low or high rate. "Warm" models initiated conversation and expressed interest by maintaining eye contact and a casual body posture, whereas "cold" models sat next to the patron without initiating or responding to conversation. Subjects exposed to a warm high-consumption model drank the most, whereas the cold model conditions did not differ significantly from each other or from the no-model control condition.

Characteristics of the model were manipulated in these initial studies. Subsequent research has also focused on the characteristics of the subject. Summarizing the evidence, Collins and Marlatt (1981) conclude that modelling has

a powerful effect wherein an individual's consumption of alcohol will vary to match that of a drinking partner.... The modeling effect is modified by characteristics of the subject including sex...and drinking history.... Heavy drinking males seem to exhibit the strongest response to a heavy drinking model of the same sex. The results of research on the effect of characteristics of the model suggests that for both male and female subjects, the consummatory behavior of a male partner is more likely to be modeled than that of a female partner. (p.235)

5.1.4 *The Determinants of Problem Drinking*

A fundamental question about alcoholism is why the alcoholic continues to drink despite serious negative consequences to his or her physical health, psychological well-being, and social function. In the same vein, why, following a period of treatment-produced abstinence, do many alcoholics suddenly revert to dangerous drinking? The disease theory of alcoholism employs the concepts of craving and loss of control in trying to answer these questions. Cogent critiques of these concepts have discredited their traditional formulation (Marlatt, 1978; Mello, 1975; Pomerleau et al., 1976; M.B. Sobell, 1978). An alternative social learning account of the phenomena encompassed by the concepts of "craving" and "loss of control" has been developed; it enjoys considerable empirical support and has proved useful in generating innovative and effective treatment methods.

5.1.4a Craving. Why does the abstinent alcoholic take that first drink?

A social learning account rejects the traditional view that drinking is an automatic, involuntary response that is forced upon the person by an overpowering physical demand for alcohol. Rather, the person desires and expects specific consequences, running the gamut from the reduction of aversive states (e.g., stress) to the attainment of positive states (e.g., sexual satisfaction) (Brown, 1985; Marlatt, 1987; Southwick et al., 1981). It is important to note that expectations about the presumed reinforcing effects of alcohol do not have to be veridical in order to influence behavior. Male alcoholics, for example, reliably anticipate that alcohol consumption will increase their sexual responsiveness when in fact alcohol produces a significant linear decreasing effect on capacity (Wilson, 1981). The abstinent alcoholic anticipates the positive or negative reinforcement that alcohol produces — or at least that he or she

believes it produces. This anticipation is important since the expectation of reinforcement may be as powerful as, if not more powerful than, actual reinforcement (Bandura, 1977). This desire for reinforcement is assumed to be labelled by the alcoholic as craving for alcohol.

In addition to expectations about alcohol's favorable effects, the alcoholic may experience conditioned reactions to environmental or emotional stimuli that have been associated with prior withdrawal states. Siegel's framework of Pavlovian conditioned compensatory responses describes how such cues might elicit craving for alcohol and subsequent loss of control over drinking (Poulos et al., 1981). This research has shown that a variety of environmental, emotional, and physiological cues can become conditioned stimuli that elicit the compensatory responses that are inevitably associated with the repetitive ingestion of alcohol. These conditioned responses may be viewed as an important part of the psychobiological substrate of craving, and even as withdrawal symptoms. Spelling out the therapeutic implications of this research, Poulos et al. (1981) suggest that

classically conditioned compensatory responses provide an important motivational component in drug use, and by extension, in drug relapse. The primary implication of a conditioning model is that treatment programs must incorporate procedures which lead to the extinction of cues associated with drug use. A client may well report little desire for a drug in an insular treatment environment, not because he is cured, but rather because there are few relevant cues to activate drug craving. When, however, the client is returned to his pretreatment environment which contains strong drug-associated cues, drug-compensatory CRs and craving would be manifest. (pp.209-210)

Ludwig and Wikler (1974) earlier advanced a similar model of craving based on classical conditioning.

Classically conditioned reactions from prior withdrawal states, elicited by cues in the abstinent alcoholic's environment, may increase the likelihood that he or she will desire a drink. However, whether or not drinking will occur will depend on a variety of factors, such as the prevailing reinforcement contingencies, as indicated below. Convincing evidence of the development and function of classically conditioned craving in problem drinkers does not exist. Improved research in this area in problem drinkers promises to yield valuable information.

5.1.4b Loss of control. According to the disease theory of alcoholism, once a drink is taken, craving is increased and the physical demand for alcohol overrides any cognitive or voluntary control (Jellinek, 1960). According to social learning theory, the consequences of one or more drinks for the abstinent alcoholic will, as is the case in any other behavior, be a function of cognitive set of the alcoholic, the social setting in which drinking occurs, and the specific reinforcement contingencies in that environment. Non-problem and problem drinking are viewed on the same continuum and governed by the same cognitive processes and laws of learning (Bandura, 1969; Marlatt, 1978).

The Jellinek (1960) notion that a small quantity of alcohol in the bloodstream precipitates involuntary drinking ("one drink, one drunk") has been disconfirmed by experimental research. Building on the research by

Engle and Williams (1972), Marlatt et al. (1973), using the balanced placebo design, had nonabstinent alcoholics and matched social drinkers sample and evaluate the taste of either alcoholic or nonalcoholic beverages during a taste-rating task. The only significant determinant of overall beverage consumption, and subjects' later estimates of the alcohol content of their respective drinks, was the expectancy factor. Regardless of the actual alcohol content of the drinks, both alcoholics and social drinkers consumed significantly more beverage if they believed they were sampling drinks containing vodka. Similarly, subjects assigned to the "told tonic" conditions consumed relatively little beverage, whether or not the drinks actually contained alcohol. In a replication of this study, employing a broader range of dependent measures, Berg et al. (1981) provided strong support for the influence of expectations on drinking by alcoholics. Berg et al. showed that alcoholics' responses to a small priming dose of alcohol were determined

by the expectancy of the drug effect rather than by the pharmacological properties of alcohol. The alcoholics showed reliable expectancy effects on the amount consumed, on rate of drinking, on time to the first sip of the last glass, on verbal responses referring to alcohol, on concurrent irrelevant motor behavior, and on situational anxiety. In addition, expectancy effects were apparent in the experimenter's rating of craving and in the psychiatrist's and nurses' ratings after the experiment. In contrast to this wide spectrum of expectancy effects, reliable effects of the actual drug were restricted to more frequent sipping with alcohol in the drink, more anxiety with alcohol and more rated intoxication by the psychiatrist. (p.63)

These results clearly demonstrate the importance of cognitive factors in regulating subsequent alcohol consumption in alcoholics following the first drink. They should not be taken to imply that the ingestion of alcohol itself is unimportant in this process. A third study in this line of research, by Stockwell et al. (1982), underscores this point. Using the balanced placebo design, with each subject serving as his own control and thus experiencing all four conditions (Berg et al., 1981, also used a repeated measures design of this kind), Stockwell et al. studied the responses of alcoholics to a priming dose of alcohol. Ten subjects were independently assessed by a psychiatrist as being severely dependent on alcohol and ten as mildly or moderately alcohol dependent. To summarize a complex but revealing pattern of findings: The severely dependent alcoholics were more likely to drink after consuming a priming dose of alcohol as opposed to a nonalcoholic beverage, irrespective of what they believed the contents of the priming dose to be. The moderately dependent alcoholics, however, showed the reverse pattern, being more influenced by the instructional set they had been given than by the actual contents of the priming dose. The significance of these results, assuming that they hold up upon replication, is obvious. The degree of physical dependence in alcoholism is shown to be a critical determinant of vulnerability to craving and loss-of-control drinking. In something of a departure from the position adopted by Marlatt et al. (1973) and Berg et al. (1981), Stockwell et al. (1982) arrived at the following conclusion:

Thus, it would seem that the priming effect of a few drinks on an alcoholic's disposition to drink is, for [severely dependent alcoholics] not merely a result of

their culturally-induced beliefs about the effects of alcohol, a self-fulfilling prophecy or a favourite rationalization for getting drink; it still occurs without the drinker being aware he has consumed alcohol. Disposition to drink alcohol would seem to have powerful psychobiological components for severely dependent people even in the artificial hospital environment in which this experiment was conducted.... [The] demonstrable importance of psychobiological aspects of alcohol dependence in no way undermines a learning model account of the syndrome. (p.521)

As a final note, these findings dovetail with the emerging consensus among clinical researchers and practitioners that degree of physical dependence is an important factor in deciding on both treatment goals and the appropriate therapeutic methods (see below).

Direct assessment of alcoholics' drinking behavior in the laboratory when they had ready access to alcohol has clearly shown that loss of control is not inevitable and that drinking is governed more by its consequences than by the mere presence of alcohol in the drinker (Cohen et al., 1971; Lawson et al., 1976; Wilson et al., 1975). In the latter study, a punishment contingency (a brief, mild electric shock to the hand) effectively suppressed drinking in chronic alcoholics even though priming doses of alcohol had ensured that they had BALs of 100 mg% or more. Similarly, Bigelow et al. (1974) showed that implementing a time-out procedure, in which 10 to 15 minutes of stimulus deprivation and social isolation were made contingent on a patient's taking a 1-ounce drink of alcohol, reduced drinking by approximately 50% of baseline frequency in nine out of the ten subjects. Drinking immediately returned to high levels with the removal of the time-out contingency. They also found that time out could suppress further drinking even after these chronic alcoholics had been consuming 12-24 ounces of 95-proof ethanol daily for four to eight days.

Alcoholics' drinking is regulated not only by its varied consequences but also by modelling influences. In a study by Caudill and Lipscomb (1980), three male alcoholics were paired with male confederates posing as alcoholics. In the first of two experiments subjects participated in a 1-hour taste-rating task (wine); two of the three alcoholics matched the drinking rate modelled by the confederate. The third subject began modelling only after the confederate had proven himself a peer by drinking heavily. In the second experiment subjects were given ad lib access to alcohol in a semi-naturalistic bar setting. The consumption rate of all three subjects matched that modelled by the confederates. The results suggest that the alcohol consumption of alcoholics can be modified by modelling, particularly if the model is seen as a peer.

Pomerleau et al. (1976) summarize much of this body of evidence as follows:

Although there were some consistencies in drinking patterns, such as a tendency toward episodic and nocturnal drinking, and a propensity for taking straight drinks in large gulps, the picture which emerged was far more complex than had been suggested previously. While the alcoholics were obviously drinking more alcohol than would social drinkers, they were not consistently drinking themselves into a stupor. The amount consumed was inversely related to the amount of work required to obtain the drinks. Some alcoholics were

observed to taper off drinking in an attempt to avoid withdrawal symptoms. Several subjects appeared to adjust their intake to correspond with that of others, a few stopping drinking altogether when a drinking partner was no longer available. Thus, when drinking behavior was observed directly, economic, physiological and social factors were found to exert a marked influence on drinking patterns. (p.852)

5.2 EVALUATION OF TREATMENT OUTCOME

As emphasized at the beginning of this chapter, the value of a theory rests not only in how well it explains laboratory findings and other research bearing on the nature of alcohol use and abuse, but also in the efficacy of the treatment methods that it generates. Social learning theorists have employed a range of treatment methods for alcoholism, as they have for other clinical disorders. For the most part, these treatment methods have been empirically evaluated with the result that some have been scrapped, others refined in the light of the available evidence, and novel strategies progressively introduced. This responsiveness to basic and applied research findings in clinical intervention has set the social learning approach off from other treatment approaches to alcoholism.

5.2.1 Aversion Therapy

In the early stages of behavior therapy, alcoholism was treated with classical aversion conditioning, in which electrical shock and chemically induced nausea served as unconditioned stimuli. Both laboratory and clinical studies have shown that electrical aversion conditioning is ineffective (Marlatt, 1973; Wilson, 1978a). Wilson and Davison (1969) argued that if aversion conditioning were to be used, animal conditioning research pointed to a more "biologically appropriate" aversive stimulus, such as nausea-inducing drugs like emetine. Drug-induced nausea had been extensively employed many years earlier by Lemere and Voegtlis (1950) in their treatment of alcoholics with aversion therapy. They reported an overall abstinence rate of 51% for all their clients over a 1-to-10-year follow-up period; 60% remained abstinent for 1 to 2 years. Consistent with these results, Wiens et al. (1976), using drug-induced nausea as an unconditioned response in an aversion therapy program modelled closely after Lemere and Voegtlis's (1950) procedures, reported an abstinence rate of 63% in a 1-year follow-up of comparable patients.

In contrast to the uncontrolled clinical reports of Lemere and Voegtlis (1950) and Wiens et al. (1976), in which the use of aversion therapy was confounded with other treatment factors, controlled research has yielded disappointing results. Cannon et al. (1981) found that although the chemical conditioning technique created an aversion to alcohol when the patients were still hospitalized, it did little to enhance the effectiveness of the basic treatment program to which it was added.

Cautela (1967) introduced the technique of covert sensitization, in which the alcoholic is instructed to imagine aversive consequences, such as nausea, in association with drinking. If aversion therapy is to be used, this technique is recommended, according to social learning theory, on several counts. First, by coupling drinking with nausea, it is theoretically consistent

with taste aversion research (Wilson & Davison, 1969). Furthermore, a social learning theorist does not even have to attribute the superiority of nausea as an aversive stimulus simply to selective biological preparedness. In terms of Bandura's (1977) social learning theory, an optimal aversive stimulus is one that can be easily recalled in imagery and cognitively rehearsed. Nausea or sickness is a natural feeling that everyone has experienced at one time or another. It is often all too easy to remember what feeling nauseated is like! Electric shock, by contrast, is an artificial stimulus that most people rarely, if ever, experience. Second, covert sensitization focuses directly on the self-activation of a reaction that social learning theory regards as a crucial process in aversive conditioning of humans. Third, it is practical since it can be implemented by the client in any setting. And fourth, it spares both the client and the therapist the unpleasant experience of the client's receiving electric shock or drug-induced nausea.

In a review, Wilson (1978a) concluded that the results with covert sensitization were disappointing. At that time the evidence consisted of positive findings from studies that were too seriously flawed to permit unambiguous interpretation (Ashem & Donner, 1968) or well-controlled analogue research that showed little specific effect of aversive imagery on alcohol consumption (Wilson & Tracey, 1976). Elkins (1980), however, demonstrated a link between the establishment of a conditioned taste aversion and successful outcome with covert sensitization. Patients who showed physiological indications of nausea when thinking about alcohol tended to retain their sobriety. Olson et al. (1981) found that a combination of covert sensitization and relaxation training added to the efficacy of an inpatient milieu therapy program up to 18 months following discharge.

5.2.2 Social Skills Training

Recently social learning theorists have emphasized the development of social and self-regulatory skills for coping with psychosocial situations that have been reliably associated with problem drinking. Marlatt's (1978) analysis of the psychosocial determinants of relapse, and his laboratory finding that heavy social drinkers consumed less alcohol when confronted with a stressful interpersonal situation if they were provided with an alternative coping strategy (Marlatt et al., 1975), as discussed above, led directly to the evaluation of social skills training with alcoholics.

Chaney et al. (1978) randomly assigned patients in a Veterans Administration hospital to the following three groups: (a) a skill training group; (b) a discussion group that focused on feelings such as anxiety and anger, which inhibit effective assertiveness; and (c) a control group that received the routine alcohol treatment program. The social skills program included modelling, therapist coaching, group feedback, behavioral rehearsal, and repeated practice, to teach each patient a variety of specific skills to cope effectively with a range of high-risk situations. Outcome measures included standardized tests of assertive behavior and a specially designed Situational Competency Test, administered both prior to and following the training program. In this test, subjects were instructed to respond verbally to a high-risk situation described by a narrator on an audiotape. Their responses were scored for a number of

components related to the quality and overall competence of the reply.

A 1-year follow-up showed that the alcoholics who had received skill training showed a significant decrease in the duration and severity of relapse episodes compared to the other two groups. In addition, performance on the posttreatment administration of the Situational Competency Test was found to be predictive of subjects' drinking behavior during the follow-up. Neither demographic data nor alcoholics' pretreatment drinking history predicted outcome. Subjects who received social skills training showed progressive improvement during the 12-month follow-up. These data support a social learning analysis of treatment effects, which predicts better functioning as people increasingly cope more effectively with situations that previously had led to drinking. Presumably, subjects improved their coping skills as the year progressed. This pattern of posttreatment functioning contrasts with the traditional findings of gradual deterioration (relapse) as a function of time from end of treatment (Marlatt, 1983).

5.2.3 Reinforcement Methods

Evidence demonstrating that alcohol use and abuse are influenced by reinforcement and punishment has been summarized above. In a direct extension of these learning principles to treatment, P.M. Miller (1975) organized a program in which community resources were provided to ten public drunkenness offenders contingent upon sobriety. During the 2 months of the experiment, this group had significantly fewer arrests for drunkenness than during the prior 2 months, and fewer than a randomly assigned control group. Similar effects were found for employment status.

5.2.4 Multifaceted Treatment Programs

The application of limited techniques (such as aversion therapy) to isolated aspects of the alcoholic's behavior has given way to more sophisticated analyses of the nature of alcohol abuse and an emphasis on multi-component social learning treatment programs. In most instances the use of these varied behavioral procedures is based on laboratory and controlled clinical studies demonstrating that they are capable of decreasing excessive alcohol consumption (W.R. Miller & Hester, 1980, 1986).

Hunt and Azrin (1973) designed a community-reinforcement program in which vocational, recreational, social, and familial reinforcers for the alcoholic were developed and made contingent on continuing sobriety. Patients were also given training in social skills and assisted in obtaining employment. Compared to the standard hospital treatment program, the community-reinforcement program produced significantly better outcomes, in terms of percentage time spent drinking, unemployment, and reinstitutionalization, over the 6-month follow-up.

These findings were replicated in a second study by Azrin (1976) that included a 2-year follow-up. The community-reinforcement treatment in this latter study was expanded to include, among other improvements, an innovative method for ensuring compliance in taking prescribed Antabuse (disulfiram), and once again proved to be significantly more effective than the control treatment. Although questions can be raised about the adequacy of the control

groups in both of these studies, methodological strengths such as the use of multiple measures of outcome, independent evaluation of success, and long-term follow-up increase one's confidence in these results.

Sobell and Sobell (1978) investigated individualized behavior therapy, in which one of the treatment goals was controlled drinking as opposed to the traditional outcome objective of total abstinence. A sample of 70 male chronic alcoholics who were inpatients at a state hospital were assigned to four different experimental conditions: a controlled drinking experimental group (CD-E), with $n = 20$; a controlled drinking control group (CD-C), with $n = 20$; a nondrinking experimental group (ND-E), with $n = 15$; and a nondrinking control group (ND-C), with $n = 15$. The two control groups received the conventional hospital treatment for alcoholics, such as large therapy groups, AA meetings, and drug therapy. The experimental groups received 17 sessions of a multifaceted behavioral treatment program in addition to the routine hospital program.

The behavior therapy sessions were devoted to making a detailed behavioral analysis of each alcoholic's drinking problem and to what the Sobells call "stimulus control" sessions. These consisted of identifying "subject-specific" setting events for each individual's drinking, training the subject to generate a series of alternative responses to those situations, evaluating each of the delineated alternatives for potential short- and long-term consequences, and then exercising the response that could be expected to incur the least self-destructive long-term consequences. To shape controlled drinking behavior, subjects were permitted to drink during sessions 4 to 16 under specific contingencies. Taking a straight drink, gulping it, or consuming more than three total drinks within the session resulted in a 1-second electric shock on a variable ratio schedule. In addition, subjects were videotaped while drunk (sessions 1 and 2) and confronted with videotaped playback of the drunken behavior in sessions 4 and 5 in order to provide feedback of their behavioral deficiencies.

Comprehensive follow-up evaluations were obtained at each 6-month interval during the first 2 years and then 3 years after the end of therapy. Estimates of daily alcohol consumption were gathered, with every attempt made to corroborate subjects' reports by securing reports from significant others in the subjects' environment who could best substantiate their reports. For purposes of evaluating the results, abstinent and controlled drinking days (days during which 6 ounces or less of 86-proof liquor or its equivalent in alcohol content were consumed) were summarized as "functioning well"; drunk days (days on which 10 or more ounces were consumed) or days during which subjects were incarcerated were summarized as "not functioning well." Both experimental groups were found to be significantly superior to their respective control groups in terms of number of days functioning well at both the 6-month and 1-year follow-up evaluations. At the 2-year mark the CD-E group was significantly different from the CD-C group. The differences between the ND-E and ND-C subjects approached but did not reach significance at both the 18-month and 2-year follow-ups. Evaluation of adjustment to interpersonal relationships and problem situations revealed the same pattern

of results as for drinking. Subjects in the CD-E group were classified as significantly more improved than CD-C group members at each follow-up over the 2-year period. Subjects in the ND-E group were rated as significantly more improved than ND-C subjects during the first year but not during the second year of follow-up. Unlike the 1- and 2-year follow-ups, which were conducted by the original investigators, the 3-year evaluation was an independently conducted, double-blind follow-up (Caddy et al., 1978). The results were consistent with those of the previous 2 years.

The limitations of this study are noted elsewhere (Rachman & Wilson, 1980). The comparison between controlled drinking and abstinence as treatment goals is confounded by assignment to the controlled drinking groups of subjects who had "requested controlled drinking, had significant outside social support for such behavior, and/or had successfully practiced social drinking at some time in the past" (Sobell & Sobell, 1973, p.54). The probability that subjects assigned to the nondrinking conditions did not have much social support in the natural environment would explain the failure of ND-E subjects to continue to show superior improvement compared to control subjects over the second year of follow-up. The maintenance and generalization of treatment effects ultimately depends on the nature of the sources of reinforcement that are available in the natural environment to support therapeutic behavior change. The strengths of the Sobells' study include the detailed, multiple measures of outcome (both alcohol consumption in particular and broader indices of personal functioning); their success in contacting well over 90% of their subjects for detailed follow-up evaluations; and long-term follow-up.

Pendery et al. (1982) reported a 10-year follow-up of the 20 subjects in the CD-E group of Sobell and Sobell's (1978) study.* Only one, whom Pendery et al. claim was not physically dependent upon alcohol at pretreatment, had maintained a pattern of controlled drinking. Eight continued to drink excessively—regularly or intermittently—despite repeated damaging consequences; six abandoned their efforts to engage in controlled drinking and became abstinent; four died from alcohol-related causes; and one, certified

*On the basis of the Pendery et al. (1982) evaluation, Maltzman publicly accused the Sobells of fraud. In response, the Addiction Research Foundation of Ontario, by whom the Sobells are employed, appointed a blue-ribbon panel of independent investigators to examine this allegation. The conclusion of this committee (Dickens, Doob, Warwick, & Winegard, 1982) reads as follows:

"The Committee has reviewed all of the allegations made against the Sobells by Pendery et al. in their Draft manuscript, in their published *Science* article, and in various statements quoted in the public media. In response to these allegations, the Committee examined both the published papers authored by the Sobells as well as a great quantity of data which formed the basis of these published reports. After isolating each of the separate allegations, the Committee examined all of the available evidence. The Committee's conclusion is clear and unequivocal: The Committee finds there to be no reasonable cause to doubt the scientific or personal integrity of either Dr. Mark Sobell or Dr. Linda Sobell" (p.109).

about a year after discharge from the research project as disabled because of drinking, was missing. Regrettably, Pendery et al. did not report follow-up data for the subjects in the CD-C group. Without this comparison standard, it is impossible to attribute these dismal long-term results either to controlled drinking or the Sobells' procedures per se.

In terms of social learning theory, the extratherapeutic environment to which clients return after treatment is a major determinant of whether or not clients maintain their treatment-produced improvement. As Moos and Finney (1983) point out: "An intervention program is but one (indeed, a temporary one) of the multiple environmental microsystems or specific settings in which a client is involved. During treatment, and even more directly afterward, a client is exposed to a myriad of influences emanating from other, more enduring microsystems such as family and work environments" (p.1040). For example, negative life events after the end of treatment predict subsequent adjustment. Accordingly, Moos and Finney (1985) stress that posttreatment factors must be taken into account in the evaluation of treatment outcome. In clear reference to the Pendery et al. (1982) argument, based on their 10-year follow-up, Moos and Finney (1985) make the following observation:

As a case in point, 6 hours of outpatient treatment may have some short-term benefit for a client, but since any such benefit is likely to be "diluted" by a client's stressful life situation, there is little reason to expect any substantial effects 4 years after treatment (Polich et al., 1981). It makes even less sense to expect strong evidence of treatment benefits 10 years after treatment (Pendery et al., 1982). These considerations highlight the need for a paradigm shift in evaluations of alcoholism programs. (p.176)

Pendery et al. also presented their own independent evaluation of the fate of the CD-E group during the first 2 years of follow-up, based on new interviews with the subjects and official (hospital and jail) records. Although Pendery et al. claim that their evaluation disputes the Sobells' 2-year data, close examination of the two accounts reveals essential agreement. What is apparent is that while the Sobells accurately reported the greater number of hospitalizations and incarcerations of the CD-E than the CD-C during the first 6 months of follow-up, they interpreted them differently. For example, they speculated that this difference "might have been the result of voluntary hospitalizations among the experimental subjects, either to curb the start of a binge or to avoid starting drinking at all" (Sobell & Sobell, 1973, pp.65-66). Pendery et al. (1982) make the case that these rehospitalizations "were not isolated setbacks in persons with otherwise benign controlled drinking outcomes. Rather, they indicated the pattern of serious problems that characterized these subjects' continued attempts to practice social drinking" (p. 173). (See Marlatt, 1983, and Sobell & Sobell, 1984, for incisive critiques of Pendery et al.'s 1982 follow-up study.)

W.R. Miller et al. (1980) used a dismantling design (see §3 above) to evaluate the components of a multifaceted behavioral program in the treatment of problem drinkers. The focus was on problem drinkers rather than alcoholics, and most of the subjects showed no evidence of physical dependence on alcohol. A total of 56 men and women were randomly assigned to the

following four groups: (a) bibliotherapy, in which clients received self-help materials but no treatment sessions; (b) behavioral self-control training (BSCT), consisting of six weekly sessions; (c) BSCT plus 12 sessions of relaxation, communication, and assertion training; or (d) BSCT plus 12 sessions of individually tailored behavioral treatment. Of these subjects, 45 began treatment and 41 completed it.

Broad assessment of outcome showed that all groups improved significantly on posttreatment drinking measures (based on subjects' self-report, self-monitoring, and information from collateral sources). The only significant difference was that the bibliotherapy group were intoxicated (BAL 80 mg%) more hours per week than the others. Miller and Taylor (1980) carried out a similar study to that of Miller et al. (1980). A self-help manual was compared to the BSCT program and to the BSCT program with relaxation training. All groups showed significant improvement, but there were no differences among the groups.

Miller and Baca (1983) reported a 2-year follow-up of the Miller et al. (1980) and Miller and Taylor (1980) studies. Of the 82 subjects, 69 were interviewed and administered breathalyser tests (all were negative). Total alcohol consumption during the 3 months preceding this assessment, as well as peak BALs during this period, were used as drinking measures. The correlation between subjects' self-reports and collateral sources was $r = .65$. The major finding was that the significant improvements obtained on drinking and other measures at prior follow-up points were found to be maintained at 2 years. More than 80% of the subjects showed improvement (abstinent or controlled drinking) at 24 months relative to any prior follow-up point. Relapse rates from controlled drinking were found to be similar to those from abstinent outcomes. Severity of problem drinking at intake remained the best single predictor of controlled drinking versus abstinent outcome, with more severe cases tending toward abstinence.

The finding that a behavioral self-control program for problem drinkers is equally effective when administered by therapists or self-administered via a detailed self-help manual has been replicated in three additional studies (Miller, 1982). The cost effectiveness of Miller's self-help approach is further documented in his finding that bibliotherapy was significantly more effective than a waiting-list control group that did not receive the treatment manual and another control group that self-monitored drinking without the manual (Miller & Taylor, 1980).

5.3 CONTROLLED DRINKING AS A TREATMENT GOAL

The alcoholism field in North America has been in an uproar during the past decade over the issue of controlled drinking (Marlatt, 1983; W.R. Miller, 1982; Pendery et al., 1982; Sobell & Sobell, 1984). A social learning or behavioral approach does *not* necessarily favor a controlled drinking goal over abstinence. Abstinence might well be the preferred goal of behavior therapy, but for reasons different from those incorporated in the disease concept of alcoholism. Nevertheless, most of the empirical research evaluating the feasibility of controlled drinking has been conducted by behavioral

researchers. Several reasons account for this development. One is that behavioral investigators have characteristically conducted innovative research on a broad range of clinical disorders; they have been the "young Turks" of modern clinical psychology. A second, more important, reason has been their theoretical framework, emphasizing as it does a continuum of drinking problems. Inherently, this model allows for the possibility of different goals for different types of drinking problems.

Clinical and experimental evidence has now firmly established that at least some alcoholics can return to controlled drinking. Some do this spontaneously following traditional treatment programs aimed at abstinence, and others, to a greater degree, following participation in therapy geared to this objective (Heather & Robertson, 1983; W.R. Miller & Hester, 1980; Polich et al., 1981; Sobell, 1978). The important question has become that of which patients are suitable for controlled drinking and which for abstinence as treatment goals. Despite the frequently furious opposition it has encountered in the United States, a decade of research on this topic has provided important data that make it possible to answer this question. Insofar as these empirical findings have useful implications for how different problem drinkers should be treated, they affirm once more the necessity of carrying out controlled research in preference to reliance on personal convictions. W.R. Miller (1982) summarizes the evidence as follows:

In general, clients who eventually succeed in moderating their drinking are those who are younger, have fewer alcohol-related life problems, have less family history of alcoholism, and show fewer signs of addiction and of medical deterioration. Those who become successful abstainers, on the other hand, show precisely the opposite characteristics (Miller & Hester, 1980). The picture that emerges is strikingly clear: the more advanced the drinking problem, the poorer the chances of achieving moderation and the greater the advisability of abstinence. With early stage problem drinkers, on the other hand, prognosis is generally better with moderation-oriented programs than in traditional abstinence-oriented methods. (p.17)

This interaction between outcome and severity of dependence on alcohol was found in two major outcome studies — the "Rand report" (Polich et al., 1981) and Orford et al.'s (1976) investigation in England. It is consistent with Hodgson et al.'s (1979) laboratory studies on the psychobiological determinants of drinking, as summarized above.

Two other studies support W.R. Miller's (1982) conclusion regarding the relative merits of abstinence and controlled drinking. Sanchez-Craig et al. (1984) compared the effects of a multifaceted cognitive-behavioral treatment with goals of either abstinence or controlled drinking in early-stage problem drinkers. Both treatment conditions produced significant reductions in alcohol consumption over a 2-year follow-up, but did not differ from each other. Sanchez-Craig et al. concluded that, for this population, controlled drinking is the appropriate outcome goal because "it was more acceptable to the majority of the clients, and most of those assigned to abstinence developed moderate drinking on their own" (p.390).

Foy et al. (1984) treated chronic alcoholics with a multifaceted cognitive-behavioral treatment program. Half of the alcoholics received training in

controlled drinking skills, half did not. During the first 6 months of follow-up, subjects in the controlled drinking condition experienced significantly fewer abstinent and more abusive-drinking days than their counterparts in the abstinence condition. These differences were no longer statistically significant during months 7-12 of follow-up, although the trends persisted. These results are different from those obtained by Sobell and Sobell (1978), a difference Foy et al. attribute to more severely dependent alcoholics in their controlled drinking condition than in the Sobells' study.

6. PRACTICAL IMPLICATIONS

The success of various therapeutic strategies and techniques associated with social learning theory, described above, clearly demonstrates the practical value of this approach in the treatment of drinking problems. This section focuses on the implications of social learning theory for prevention of alcohol abuse.

The need for effective education of the public about alcohol's effects on health and behavior is generally accepted. The content of public education about alcohol must be firmly based on the best available knowledge and conveyed effectively. Unfortunately, many educational efforts have been ineffective or counterproductive because of problems in either content or form (Nathan, 1983). Since social learning research has contributed importantly to our knowledge of alcohol's effects on emotion and social behavior, it inevitably has implications for what the public at large should be told. Even more important, however, are its implications for the way in which information about alcohol should be disseminated. To illustrate this point, consider the manner in which alcohol and drinking are portrayed in the media, particularly television.

Television's influence on our daily lives is well documented (e.g., Rubenstein, 1983). From a social learning viewpoint, with its emphasis on vicarious learning, this influence is axiomatic. Hence, television has enormous potential for affecting attitudes towards alcohol and actual drinking behavior. Singer (1983) points out that "children spend more time in the U.S. watching television than they ever spend in school" (p.815). Citing the 1982 NIMH report "Television and Behavior," Senator Heinz (1983) draws attention to the fact that "prime-time television programs watched by children present each year about 3,000 instances of drinking alcoholic beverages. Should we be surprised that one of this nation's greatest health problems is alcohol abuse?" (p.817).

Empirical research on the specific impact of television on drinking habits is minimal, as Collins and Marlatt (1981) note. They do, however, refer to Lowery's (1980) unsurprising finding that drinking is portrayed as a major positive reinforcement with no deleterious consequences. Indeed, it is hard to dispute Mosher and Wallack's contention (cited in Bonnie, 1981) that media advertising "creates the impression that drinking increases sexual prowess, promotes social acceptance and success, insures pleasure without harm, and

helps solve personal problems" (p.167). Bonnie (1981) observes that the government "has not revised its alcohol advertising regulations since they were first promulgated in 1935. [It] has, in effect, deferred to the self-regulation of the alcohol beverage industry and the National Association of Broadcasters. By voluntary action, the National Association of Broadcasters (and the liquor industry) have agreed to ban broadcast advertising of hard liquor and to restrict beer advertising to certain hours" (p.167). Yet problems with advertising persist.

Take beer advertising, for example. *Consumer Reports* (1983) notes that the "money spent on beer advertising in almost all media rose from \$92.7-million in 1972 to \$406.5-million in 1982 — an increase of 338 percent" (p.348). The companies show a keen appreciation of social learning principles as well as the power of the dollar. The Miller Brewing Company has used former athletes as influential models drinking Miller Lite. As *Consumer Reports* notes, "if drinking Lite beer was the thing for Mickey Mantle, then drinking Lite must be okay for everyone else." And, of course, it works. The success of Lite beer, the magazine continues, "represents the ultimate achievement of advertising — being able to sell a lower-cost product at a higher price" (p.349). Miller has also shifted attention from the product itself to its reinforcing consequences. "Miller time" is presented as the just reward for a hard day's work. On New York television, Anheuser-Busch (Budweiser) systematically pairs advertisements for its beer with every home run that a Yankee baseball player hits.

Advertising is targeted at young drinkers, particularly the large college audience, even though many in that audience are still below the legal drinking age. Techniques include heavy advertising in college newspapers, sponsorship of campus events, and free distribution of beer ("reinforcer sampling" in the argot of operant conditioning).

The message is not confined to advertising, but pervades other heavily watched television fare. In *Dallas*, the top-ranked program on commercial television in the U.S. for many years, J.R. Ewing can frequently be seen reaching for a drink (spirits, without mixers), at any time, in any place (including his office). In one scene, following a stressful family conflict, J.R. takes a glass of champagne only to have his macho father declare that "I need something stronger than that" — to cope with the stress, one assumes. Here drinking for the most dangerous reasons is modelled and given a verbal label, thereby enhancing an already powerful modelling effect.

The point of these illustrations is that modelling and other social learning principles are an integral part — deliberate in the case of beer advertising, unwitting, presumably, in the *Dallas* example — of the media. At present, this modelling almost certainly works against the development of safe and sensible drinking patterns. The situation nevertheless can be changed. The same influences can be used to encourage moderate drinking under appropriate situational constraints as part of a proactive public health policy based on social learning principles. For example, Marlatt (1985) compared a skills training program, based on social learning theory, with an alcohol information program and with a no-treatment control condition in a controlled evaluation of secondary prevention methods for high-risk drinkers in a university. The skills

training program was significantly more effective in modifying both the amount and pattern of alcohol consumption.

7. RELATIONS TO OTHER THEORIES

7.1 THE DISEASE CONCEPT OF ALCOHOLISM

Social learning theory is typically pitted against the traditional disease theory of alcoholism. The latter has fared poorly in experimental evaluations of its key assumptions, as described above. For instance, Jellinek's (1960) notion of loss-of-control drinking has been discredited, whereas social learning provides a theoretically consistent and empirically supported explanation of phenomena such as craving, sudden relapse following periods of abstinence, and the well-documented return to controlled drinking in problem drinkers and some alcoholics.

Another tenet of disease theory is that alcoholism is a progressive, irreversible process in which problems continuously accumulate over time (Jellinek, 1960). Cahalan's (1978) longitudinal studies of problem drinkers dispute this notion. Based on his research, Cahalan concluded that the

prodromal status of specific drinking problems as predictors of later and more serious problems has been considerably overrated, since our general-population change data suggest that an "early warning" prediction based on such "symptoms" will yield a substantial number of false positives who will never get into serious trouble.... [We found that the] continuity of *specific* problems over time is rather low, but that the probability of future involvement in *some* alcohol problems — but not necessarily the same ones — is increased if one develops alcohol-related problems. Thus, the "progressive disease" concept of problem drinking is open to question. However, the fact that those who have drinking problems at one time tend to have drinking problems of varying kinds at a subsequent time may imply that environmental factors may play a considerable part in determining the type of alcohol-related problems that may occur. (p.246)

Similarly, Vaillant (1983) found that over the long term, alcoholism is "a self-limiting illness."

7.2 GENETIC THEORY

Alcoholism runs in families, a phenomenon that is explained by both genetic influences and social learning experiences. The evidence points to a genetic predisposition for alcoholism in many individuals (Goodwin, 1976). The evidence on genetic determination of alcoholism has led to efforts to find behavioral or biological markers of vulnerability to alcoholism in individuals at risk for the disorder. Although the mechanisms by which an inherited tendency for alcoholism ends up being expressed have not been established, individuals with a family history have been shown to differ from matched controls with no family history on a variety of behavioral and biological dimensions. There is evidence, for example, that individuals with a positive family history for alcoholism might be especially prone to develop tolerance and physiological

dependence (Schuckit, 1984). Others might be protected against the development of tolerance and dependence because they experience early aversive effects of alcohol consumption. Baker and Cannon (1982) speculate that alcoholics are less susceptible than nonalcoholics to alcohol-induced taste aversions. Asians, who have relatively low rates of alcoholism, show greater physical sensitivity to and discomfort from alcohol than Caucasians, and there is evidence linking alcoholism with an inherited insensitivity to alcohol's effects (Schuckit, 1984). Innate tolerance to alcohol of this kind could act as a moderator variable in a social learning account of the development of drinking problems, or it might be consistent with Goodwin's (1976) "either/or" concept of alcoholism.

Baker and Cannon (1982) attempt to explain why taste aversion learning does not stop alcoholics before they suffer damaging consequences. It might also be that individuals at risk for alcoholism derive greater reinforcing effects from alcohol than their less susceptible counterparts. A well-controlled study by Sher and Levenson (1982) provides a particularly revealing example of the role of individual differences in mediating alcohol's effects. Among healthy male social drinkers, subjects considered to be at increased risk for alcoholism on the basis of high scores on the MacAndrew (1965) scale were found to show significantly greater reductions in their cardiovascular and emotional responses to stress after consuming alcohol than did their low-risk counterparts. As the authors point out, not only was this difference statistically significant, but also, for all practical purposes, the low-risk subjects did not show a reduction of tension. Sher and Levenson interpret their findings to indicate that "the outgoing, aggressive, impulsive, and antisocial individuals who are identified by these measures (and who have been shown to have a high incidence of alcoholism in prospective studies) may find alcohol consumption particularly reinforcing by virtue of their obtaining a greater amount of alcohol's stress-response-dampening (SRD) effect when they drink" (p.350).

Behavioristic accounts of alcohol addiction have eschewed explanations that focus on the pharmacological properties of alcohol or something inside the person in favor of behavior-environment interactions. By introducing the role of person variables in psychological functioning, social learning theory has sought to remedy the environmental bias of behavioristic accounts. Nevertheless, it has not embraced personality differences in analysing the etiology and treatment of clinical disorders. Once again, however, either innate or acquired individual differences of the sort described by Sher and Levenson (1983) might complement a social learning account by moderating the reinforcing potential of alcohol in response to stress.

Although several studies have found strong evidence of the role of heredity in some alcoholics, they have also uncovered evidence of the critical importance of sociocultural influences. For example, in some investigations (e.g., Bohman et al.'s 1981 and Cloninger et al.'s 1981 studies), a large percentage of identified alcohol abusers had no family history of alcohol abuse — as many as 60% in the Cloninger et al. study. Additional evidence that both heredity and social learning are determinants of alcoholism comes from rare longitudinal research, which is a powerful tool for exploring the etiology of

a disorder. Vaillant (1983) has analysed three longitudinal studies, comprising 700 men and women, 200 of whom ultimately were diagnosed as alcoholic, to examine the natural history of alcohol abuse. The individuals in these different studies were assessed at regular intervals on a wide range of measures of psychological adjustment, physical health, social and economic status, and alcohol use. On the basis of this information, which he claims "no other published study [on alcohol use and abuse] can match" (p.2), Vaillant found three premorbid differences between alcoholics and non-problem drinkers: (a) alcoholics are "more likely to come from ethnic groups that tolerate adult drunkenness but that discourage children and adolescents from learning safe drinking habits" (p.311); (b) alcoholics are also more likely to be related to other alcoholics; and (c) alcoholics are "more likely to be premorbidly antisocial" (p.311).

7.3 HULL'S SELF-AWARENESS MODEL

In contrast to Jellinek's (1960) disease theory and contemporary genetic theory (Goodwin, 1976), Hull (1981) has advanced a self-awareness model of alcohol use and abuse that draws on experimental social psychology and hence is based on the same discipline as social learning theory. Hull's thesis is that alcohol "disinhibits" social behaviors by virtue of reducing an individual's level of self-awareness. More specifically, alcohol is said to interfere with

encoding processes fundamental to a state of self-awareness, thereby decreasing the individual's sensitivity to both the self-relevance of cues regarding appropriate forms of behavior and the self-evaluative nature of feedback about past behaviors. Insofar as the latter form of information can provide a source of self-criticism and negative affect, alcohol as an inhibitor of self-aware processing is proposed to provide a source of psychological relief. (p.586)

Wilson (1983) has argued that although Hull's self-awareness theory overlaps with social learning theory, the latter is more comprehensive and useful in a practical sense since it accounts for findings on alcohol's effects that either create problems for Hull's model or are not addressed by the model; moreover, it is part of a broader theory of self-regulation of behavior that has been successfully related to the clinical treatment of substance abuse and other disorders. (But see Hull and Reilly, 1983, for vigorous dissent.)

Hull points out that his model is similar to the tension reduction theory of alcohol's effects. He emphasizes, however, that in terms of his model, "alcohol does not reduce tension directly [via a pharmacological effect as alleged by the tension reduction theory] but rather serves to reduce cognizance of a potential source of tension. Thus, its primary personal effects are cognitive rather than affective-motivational" (p.589). Both theories, then, hold that people are likely to drink to avoid or escape distress. An advantage of Hull's theorizing is that it leads to the study of an individual difference (self-consciousness) that might moderate alcohol's stress-reducing function. Hull et al. (1986) reported that of treated alcoholics, those who were high in dispositional self-consciousness and who encountered negative life events during follow-up were more likely to return to drinking than their counterparts with low self-consciousness. Replication of the role of self-consciousness as a risk factor for relapse would have important implications. Although it does not address the putative advantages

of taking this individual difference into account, social learning subsumes the cognitive effect on stress that Hull emphasizes as one part of its dual-process conception of eliciting and reducing stress (see §1). The latter accommodates the cognitive mediation of stress reduction via alcohol, but also the evidence that alcohol can modify affective responses via non-cognitive routes.

Specifically, Wilson (1983) has argued that Hull's theory fails both to take account of the effects of cognitive set (expectations) on alcohol's effects, and to deal with two findings: that alcohol does not necessarily reduce self-awareness (Frankenstein & Wilson, 1984); and that alcohol consumption may produce self-evaluations in the absence of effects on self-awareness (Yankovsky et al., 1986). A social learning analysis both allows for greater independence between the self-awareness and self-evaluation components of self-regulation, and provides a more detailed analysis of the cognitive processes that lead to and define self-evaluation (Wilson, 1983).

7.4 SOCIAL LEARNING VERSUS TRAIT THEORY OF PERSONALITY

The interdependence among cognitive, behavioral, and environmental influences on psychological functioning in social learning theory makes for a theory of personality that captures the interaction between the person and the environment. The traditional behavioristic approach, with its emphasis on the overriding importance of environmental control of behavior (Skinner, 1953), has been criticized for losing sight of the role of the person — and thus lacking a theory of personality. The objection from humanistic and other psychologists has been that behaviorists treat people as though they were controlled only by situational forces rather than being free, self-directed agents responsible for their own growth and actions. Social learning theory provides a solution to this clash between two extreme viewpoints in recognizing that the characteristics of the environment interact with the nature of the people in it, and both common sense and experimental findings show how unwise it is to ignore either side of this crucial interaction.

Much debate has centred on the question of whether the person or the situation is more important in predicting behavior. This question is misguided and unanswerable. In terms of a social learning analysis, the relative importance of individual differences and situations will depend on the situation selected, the type of behavior assessed, the particular individual differences sampled, and the purpose of the assessment (Mischel, 1973). Consider the varying influence of different situations on behavior. Evidence clearly shows that an individual's behavioral patterns are generally stable and consistent over time. Nevertheless, studies also reveal that behavioral patterns are not highly generalized in different situations. The specificity or discriminativeness of behavior in different situations poses a significant problem for trait theories of personality. The central assumption of such theories is that people possess stable and generalized personality traits that determine behavioral consistency across a wide variety of different situations. Yet as Mischel (1968) has pointed out, with the exception of activities closely related to intelligence and certain forms of problem-solving, the correlations between different measures of the same trait are usually very low and there is little consistency in behavior

patterns across different stimulus situations.

Social learning theory readily accounts for the discriminativeness of human behavior. On the basis of the learning principles of reinforcement, discrimination, stimulus generalization, and cognitive person variables, a person would be predicted to act consistently across situations only to the extent that similar behavior leads, or is expected to lead, to similar consequences across those conditions. Since it is rare to find the same behavior reinforced across situations, it is not surprising that people make subtle discriminations and behave differently in different settings.

Trait theories emphasize differences among people on some dimension selected by the clinician. For some purposes, such as gross screening (e.g., administering an MMPI to a client to explore further the extent of his or her psychopathology) or group comparisons, a trait approach is useful. But it does not aid the therapist in making treatment decisions about a particular individual (Bandura, 1969; Mischel, 1968; Rachman & Wilson, 1980). Social learning theory, however, provides a powerful framework for devising behavior change strategies. It is person-centred and focuses on describing the individual in relation to the particular psychological conditions of that individual's life. The heart of this approach is a functional analysis that investigates covariations between changes in the individual and changes in the conditions of his or her life. The interest here is not in how people compare to others, but in how they can move closer to their own goals and ideals if they change their behavior in specific ways, as they interact with the significant people in their lives. In this sense, the social learning approach captures the richness and uniqueness of individual people.

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8. SYSTEMS THEORY AND ALCOHOLISM

Sheldon Pearlman

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1. PRINCIPAL ELEMENTS OF THE THEORY

Systems theory and family therapy have been intimately connected since the early 1950s, when new therapeutic modalities emerged that called for the involvement of entire families in the treatment of difficulties that had previously been treated by means of therapy. The terms "systems theory" and "family treatment" have often been used interchangeably as if they were synonymous. In fact, however, the two are fundamentally different. Systems theory constitutes a unique conceptual framework or epistemology that focuses on pattern, form, organization, and wholeness (Keeney, 1982; von Bertalanffy, 1968). Family therapy, on the other hand, refers to a wide variety of treatment methods, techniques, and interventions in which family members play a more or a less active role along with the individual manifesting problematic behavior, the latter typically being referred to as the "identified patient." In short, not all family therapy is a reflection of the application of a systems perspective and framework, and conversely, systems theory is not restricted to clinical approaches involving the family.

Interventions with entire families, marital dyads, other familial subsystems and systems extending well beyond the nuclear family may all represent clinical applications or adaptations of systems theory. While most of the references and illustrations used in this chapter will relate to the family, it being the interactional system most typically involved in and associated with systems-based treatment interventions, the reader is cautioned against restricting the relevance of systems theory to clinical interventions involving only the family.

1.1 CONCEPTUAL BASIS OF SYSTEMS THEORY

The essential premise of systems theory is that behavior is determined and maintained by the ongoing dynamics and demands of the key interpersonal system(s) within which the individual interacts, rather than by idiosyncratic, intrapsychic factors. Within this framework, the thrust is away from the view of people as primarily psychological beings, a view that has hitherto dominated the field of psychotherapy, and towards a view of people as essentially social beings. Behavior is thus conceptualized as being less a reflection of unique personality and psychological variables, and more a response to the vicissitudes of interpersonal, interactional systems. Obviously, the family is seen as a critically important system, albeit not the only one, impacting on the individual.

The term “system” is used to refer to a complex hierarchical organization of interacting, interdependent elements characterized by stable, predictable patterns of relationships between and among the elements. Within a system, a change in one element leads to compensatory changes or reactions in all elements, as well as in the system as a whole (von Bertalanffy, 1968). A systems perspective emphasizes “wholeness” and the interdependence of elements — their pattern, form, and organization — rather than the elements themselves. Steinglass (1978) suggests that this apparently simple and straightforward emphasis on “wholeness” and the pattern of interactions among the constituent elements of a system is, in fact, the most distinctive concept within systems theory.

Systems theory, when translated into clinical practice, has given rise to a number of important new concepts. The first of these is the notion of organization. Organization refers to the consistent nature of the relationships between and among the elements or subsystems that together comprise the larger system. Implicit in this notion of organization are the concepts of boundaries, differentiation, hierarchies, and non-summativity. Boundaries distinguish those elements contained within the system from other elements within the broader environment. Boundaries are significant within a systems framework since they not only define membership within a given system or subsystem but also characterize the quality of the relationship between the system per se and its surrounding milieu. This latter property of a boundary is referred to as its permeability and describes the ease of exchange of information with other systems.

Differentiation refers to the unique contributions that the various subsystems provide to the ongoing functioning of the entire system. Each subsystem fulfills a specialized role and set of responsibilities. Within a family system, for instance, one can readily contrast the differential inputs and responsibilities of the marital dyad with those of the children. These differences in roles and responsibilities among the subsystems yield a hierarchy that is reflected in such variables as complexity, power, control, and authority.

Non-summativity refers to the proposition that a system is more than the sum of its constituent parts. Simply adding up the various components of the system will not yield an accurate picture of the overall dynamics, properties, or quality of the larger system. The quality of a family’s interactional behavior, for

instance, is more than and distinct from the sum of the personalities of its members. An important implication of this principle is that the system cannot be fully understood or explained by analysing its component parts. The family takes on a "life of its own" and becomes the essential unit of analysis and focus of change.

Another important concept within systems theory is its formulation of causality. Within a systems framework, causality is viewed as being circular rather than linear. Reductionistic notions of a cause-effect relationship are replaced with more complex formulations involving feedback loops and interaction. Thus, most behavior that is observed in a family context is the result of the interaction between the relevant family members, rather than being the result of a single factor that can be isolated as "the cause." Furthermore, this behavior, in turn, is seen as the stimulus for further action and interaction, thereby contributing to the blurring of any simple cause-effect analysis. The issue of "punctuation" becomes significant within this context. Punctuation refers to the imposition by an observer of a purported behavioral sequence. The order one arbitrarily imposes on a sequence of communication or behavior may strongly influence the importance attached to each element in a causal analysis. As Keeney (1982) points out, punctuation is an attempt on the part of an observer, perhaps a therapist, to impose a meaning or explanation onto a series of related events. A simple example, encountered by all therapists working with couples in which one spouse has an alcohol-related problem, will illustrate this. When confronted by the therapist as to why he drinks, a husband comments that he drinks because his wife nags him. The wife, on the other hand, counters with the equally plausible explanation that she only nags because he drinks. If one were intent on developing a linear cause-effect hypothesis concerning the relationship between the husband's drinking and the wife's nagging, punctuation would be necessary to impose a chronological order on the two behaviors.

A systems analysis, however, would adopt a circular notion of causality and abandon the attempt to resolve, to the satisfaction of both spouses, whether the drinking caused the nagging or the nagging caused the drinking. Instead, the couple's characteristic pattern of interacting becomes the focus of attention, with both drinking and nagging being seen as the undesired by-products of that pattern. Emphasis would be placed on interactional patterns, rather than on an arbitrarily imposed sequence that defines one act as the cause of the other.

Another defining characteristic of a system is that it is a rule-governed set of interactions. Rules, whether implicit or explicit, define the essential parameters of interactive behavior within the system. In effect, rules are the mechanism whereby the order, predictability, and consistency that characterize human interactional systems such as the family emerge. Rules prescribe specific behaviors or responses and thus limit the range of options available to each system member and to the system as a whole. Virtually every aspect of interactional behavior is governed by rules. For instance, in many marriages rules emerge that define which partner initiates sexual intimacy and under what conditions, even though such initiatives are typically assumed to be

totally spontaneous. It is the existence of rules that makes the system stable, predictable, self-correcting, and capable of accommodating change.

The concept of "homeostasis" has long been used to describe a state of equilibrium or balance in living systems (*Encyclopaedia Britannica*, 1975; Steinglass, 1978). Jackson (1968) used the analogy of a thermostat that regulates and minimizes fluctuations in temperatures to illustrate the notion of homeostatic mechanisms operating within a family. Therapists have relied on the concept of homeostasis to explain the family's tenacity in holding onto existing behavioral repertoires, resisting change, and exerting pressure to minimize or reverse change when it occurs. It is this property that leads systems theorists to stress the importance of effecting change within the entire system in order to make possible and accommodate change in one of its members. In systems terms, this is referred to as helping the family achieve and maintain a new level of homeostatic functioning.

Homeostatic mechanisms act to ensure that change is introduced in a way that is tolerable to the ongoing functioning of the system, often by introducing compensatory changes to minimize disruption and disequilibrium. The mechanisms contributing to these self-regulatory processes are referred to as feedback loops. Positive feedback loops introduce change into the system. Negative feedback loops, on the other hand, promote a steady state and diminish the impact of change that is introduced into the system. Negative feedback loops are, therefore, closely associated with a system's homeostatic tendencies, and have become an important focal point for the systems therapist in attempting to identify and ultimately overcome a family's seemingly inherent tendency to resist change.

Two important and often confused aspects of homeostasis must be stressed. The first is that the notion of homeostasis does not limit or restrict a system to a fixed, immutable pattern of functioning. Homeostasis is not incompatible with theories of change. Rather, by virtue of homeostatic tendencies, a system ensures that change will be introduced in a gradual, orderly, tolerable way, with a minimum of disruption. Secondly, although homeostasis is often used in conjunction with concepts such as equilibrium, balance, and steady-state, it is important to differentiate between stability and functionality. In systems theory, homeostasis refers to the stability of current patterns of behavior or transactions, with no implied qualitative connotations concerning those current patterns. For families in therapy, this existing equilibrium clearly may be, and typically is, associated with dysfunctional patterns of interactions.

The concept of equifinality asserts that one can observe the characteristic patterns of interactions and transactions of the system regardless of the point of entry into that system. A family's typical pattern of transactions transcends the discussion of any specific issue or theme and becomes readily apparent as the family attempts to resolve problems, make decisions, or simply interact in the presence of the therapist.

Finally, information may be thought of as constituting the energizing component of human systems. The exchange of information between or among individuals is defined as communication, and includes both verbal and non-verbal processes. Following the work of Watzlawick and his colleagues (1967),

the terms communication and behavior came to be used interchangeably, since all behavior was deemed to have both implicit and explicit message value. Concern with a family's communication patterns and difficulties became a central feature of the evolution and development of systems theory, as that theory was applied to work with families. An elaborate set of axioms, or principles, of communication (often mistakenly referred to as a theory) was developed and carefully applied to the study and treatment of dysfunctional families. A review of these axioms is beyond the scope of this chapter; the interested reader is referred to Jackson (1968), Satir (1967), Sluzki and Ransom (1976), Stein (1973), and Watzlawick et al. (1967).

1.2 CURRENT STATUS OF SYSTEMS THEORY APPROACHES

Systems theory approaches to clinical intervention with dysfunctional families are increasing in sophistication and maturity. The early systems literature was dominated by attempts to differentiate, at both a philosophical and clinical level, systems-oriented approaches from psychodynamically oriented approaches. The work of the Group for the Advancement of Psychiatry (1970) stands out in this regard. Their report identified a unidimensional continuum along which family therapists could be placed in terms of self-reported adherence to a "pure" systems approach, on the one hand, or reliance on psychodynamic principles and theories in their work with families, on the other. Position Z therapists purportedly rely exclusively on a systems orientation in their work with families. Such therapists refer to the individual family member with the presenting problem as the "identified patient" and proceed to reconceptualize the problem as a family or interactional problem, with the family being defined as the appropriate unit of treatment. These therapists typically emphasize present interaction, avoid any detailed history taking, and focus on overt behaviors and interactions, rather than on the subjective experiences of individual family members. Position A therapists, on the other hand, see the family as a potentially important and useful source of leverage in the primary task of effecting change in the individual manifesting pathological behavior. Involving the family is seen as an adjunctive therapeutic tactic or technique rather than as a radically different treatment approach. In effect, the family may be conceptualized as a source of stress, tension, or assistance in terms of its impact on the individual. As Guerin (1976) pointed out, this simple classification of theoretical orientations along a single dimension of individually based psychodynamic theory versus systems theory proved inadequate as new adaptations, orientations, and schools of systems theory approaches emerged in the 1970s.

One of the dominant themes in the current systems literature is the attempt to develop new, more sophisticated and refined classifications of the various systems approaches. The focus is now on the differences *between* various systems models rather than on distinguishing between systems and non-systems approaches. The work of Levant (1980), Ritterman (1977), and Vieland (1982) reflects the ongoing attempt to develop new paradigms for purposes of classifying the various schools or approaches within the field of systems theory. Such work often takes on a highly esoteric, philosophical, and

at times parochial and polemical quality, as one school is contrasted with another or as one classification schema is advanced as an improvement over its predecessors. For instance, Ritterman's schema (Ritterman, 1977) dichotomizes systems approaches into the organismic and mechanistic world views, with the primary distinction being based on the pretheoretical assumptions or epistemology from which each approach is derived. Vieland (1982), in introducing yet another classification schema to the systems field, comments on the continuing proliferation of such schemata in spite of the fact that such efforts have been universally unsuccessful in terms of diminishing the sense of chaos resulting from a plethora of competing theories, each with its own charismatic spokesperson.

The two dominant schools or approaches within the systems field are the strategic and the structural. Both approaches emphasize the interpersonal context within which behavior occurs and both conceptualize behavioral difficulties as being system-maintained and system-maintaining (Stanton, 1981a). Each emphasizes interactional patterns and processes in the "here and now," with the therapist playing a very active, directive role. The goal of both therapies is to effect systems-level change to make possible and accommodate specific changes related to the presenting problem that prompted the family to seek treatment. Problems are typically re-defined into interactional or systems terms and the family is defined as the patient or client.

In spite of these similarities, there are some important differences.

1.2.1 *Strategic Approaches*

Strategic therapy has its roots in the work of the Mental Research Institute in California and is associated with the pioneering work of Jackson, Watzlawick, Weakland, Bodin, Sluzki, Haley, and Satir, and, more recently, with the Institute for Family Study in Milan (see Bodin, 1981). Within the strategic model, the emphasis is on communications and the sequencing of behaviors (Steinglass, 1978). Problems are conceptualized as dysfunctional communication or interactional patterns repeated by the family over the course of time, as the family attempts to deal with a myriad of difficulties. Particular attention is paid to the amelioration of the presenting problem, with the expectation that this will represent the initial step in helping the family develop a new, more adaptive level of functioning.

The family's persistence in adhering to dysfunctional behavioral patterns and its resistance to the efforts of the therapist to induce change have given rise to the use of paradoxical interventions within the strategic model. The Milan group in particular has used paradoxical methods extensively in work with families (Palazzoli-Selvini et al., 1978). Through the use of paradoxical interventions, the therapist attempts to gain a measure of control in situations where resistance is particularly well entrenched. Perhaps the most common type of such intervention is prescribing the symptom, a technique by which the therapist instructs the family to persist in the very behavior that is to be eliminated (e.g., continue arguing without attempting to reach a compromise). In such situations, the family either resists the instructions of the therapist and consequently improves (i.e., stops fighting), or complies, thereby appearing to do the therapist's bidding and acknowledging his or her authority (Stanton,

1981b). Another prominent form of paradoxical intervention involves the use of positive interpretation or connotation, by which the therapist redefines problematic behaviors in positive terms or ascribes positive motives to the individual whose behavior the family seeks to change. Thus, a therapist may express the view that a mother's overprotectiveness of her daughter is a laudable expression of maternal love and devotion and that the daughter should consider herself fortunate to have a mother who takes such an active and involved interest in her well-being.

1.2.2 Structural Approaches

Structural approaches are associated with Minuchin and his colleagues at the Philadelphia Child Guidance Clinic (Minuchin, 1974). Spatial rather than temporal patterning is the primary concern within this school, with emphasis being placed on the organizational relationships of the parts to each other and to the whole (Aponte & Van Deusen, 1981). Attention is paid to such structural aspects of the family's functioning as hierarchies, alliances, coalitions, and boundaries. A family's boundaries determine the intimacy, involvement, and distance between and among the various subsystems, with enmeshment and disengagement representing the extremes (Stanton, 1981a). Therapy is geared toward "restructuring" the family system, rather than resolving the presenting problem, with great emphasis being placed on effecting change within the treatment session itself. The therapist may "join" a system by adopting the family's communication style and practice in order to signal respect for and acceptance of the family, to lower resistance, and to enhance her or his influence as a change agent. The structural therapist, however, maintains independence and objectivity in all dealings with the family by ensuring that all such intervention techniques are consistent with the overriding goal of effecting a structural rebalancing of the family system.

1.3 SUMMARY

In summary, systems theory has increasingly become identified with and assimilated by the burgeoning family therapy movement. However, it is important to differentiate between systems theory, which represents a distinct epistemology, and family therapy. The latter represents a number of diverse clinical and theoretical approaches similar only in that the family of the individual is expected to be involved to varying degrees in treatment sessions. Many family therapy approaches are not, in fact, based on systems theory at all, but rather represent adaptations or modifications of other theoretical models to allow for the inclusion of the family. Systems theory can be applied not only to work with families but also to interventions with familial subsystems, especially the marital dyad, as well as to extra-familial contexts. Shain and Groeneveld's (1980) description of the environmental model of employee assistance programming, for example, is essentially a systems perspective applied to the workplace.

Steinglass's (1977) definition of a systems theory approach to family therapy captures the essential and distinctive elements of this perspective:

The emphasis is on patterns of interrelationships between...component parts, hence the focus on interactional behavior, structural patterning within the

family, and the balance or stability of the system as a whole. Any single piece of behavior for the family systems therapist has to be understood first in terms of how all the component parts are contributing to or making the behavior possible, and secondly, how the behavior is affecting all the individuals in the family. Pathology becomes redefined as a structural or functional imbalance in the family.... (p. 263)

Two dominant schools of family systems approaches have emerged. These are the strategic, focusing on communication and behavioral sequencing or patterns, and the structural, with its emphasis on spatial and organizational dynamics.

2. HISTORICAL DEVELOPMENT OF THE THEORY

Concern with the role that interactional family dynamics might play in the etiology, persistence, and treatment of an individual's problems clearly antedates the formal development of systems theory. As early as 1909, for instance, Freud (1964) described the treatment of a phobic boy by means of an exchange of letters between himself and the boy's father. Similarly, throughout the 1920s and 1930s the Child Guidance Movement developed in child care and family agencies. This movement was characterized by attempts to work directly with parents and often with the entire family in order to redress the school and other psychosocial difficulties experienced by children. By the 1950s, family therapy had, in fact, become a pervasive treatment modality, especially within social work. The theoretical and conceptual bases for such interventions, however, continued to be an adaptation of more traditional psychodynamic and ego-psychological theories.

It was within this context of an increasingly widespread application of a new therapeutic modality in the absence of a unique theoretical foundation for such a development that systems theory emerged. In effect, the theory, to a significant degree, emerged after the increased interest in and concern with the family among clinicians, rather than as a precursor to the evolution of family-based intervention strategies. Practitioners adopted the new "theory" to explain and rationalize trends that had already emerged in their clinical practice. Gurman's (1978) observation that the conceptual history of marital therapy constituted a "technique in search of a theory" seems relevant to this period in the development of family therapy as well. Olson (1975) and Haley (1971) echo this same observation of family therapists struggling to find a theory to fit their emerging practice.

Within this context of pragmatic eclecticism, proponents of systems theory found a ready group of potential adherents. Systems theory provided a comprehensive view of behavior, behavior disorders, and their treatment that held out the promise of bringing order, coherence, and theoretical respectability to the family therapy field.

As is the case with most theories of psychopathology currently applied to the field of alcoholism, systems theory was initially explored and developed in response to other dysfunctional behaviors. The adaptation of systems theory to

the field of psychotherapy, for instance, can be traced back to the early 1950s and the pioneering work conducted at the Mental Research Institute in Palo Alto under the direction of Gregory Bateson (see Bodin, 1981). Bateson and his colleagues, including Don Jackson, Jay Haley, William Fry, John Weakland, and Virginia Satir, were primarily concerned with examining the impact of dysfunctional family communication on the etiology of schizophrenia. Theories invoking rules, homeostasis, feedback loops, the "double bind," and the pragmatics of communication emerged from these efforts, along with attempts to more systematically study and measure family dynamics. Research was extended quickly from the initial focus on schizophrenia to an examination of how family dynamics and interactional patterns affected other conditions, including asthma and ulcerative colitis (Bodin, 1981).

At about the same time, Nathan Ackerman (1966) founded the Family Institute of New York to promote the study and treatment of the family system and became a prominent advocate and theoretician for family therapy. In 1962, the journal *Family Process* was first published and quickly became the leading journal dealing with family treatment and, later, family research.

From these beginnings, family therapy, based in some cases on systems theory, quickly grew in popularity among clinicians. Psychiatrists and social workers, in particular, seemed most eager and ready to broaden their theoretical orientation and practice to incorporate systems concepts and treatment techniques. For the most part, however, the use of systems-based family treatment was restricted to those conditions or difficulties that readily lent themselves to an interpersonal analytical framework. For instance, marital therapists, focusing on the dysfunctional and discordant interaction between spouses, quickly assimilated systems theory. Similarly, therapists working primarily with children's behavioral difficulties were among the first to apply a systems perspective to clinical practice, since the relationship between such difficulties and ongoing family dynamics seemed relatively straightforward and direct. However, the application or extension of systems-based therapies to problems with less obvious interactional ramifications proceeded more slowly.

2.1 THE APPLICATION OF SYSTEMS THEORY TO ALCOHOLISM

As recently as 1976, Steinglass commented on the relative lack of interest in alcoholism among family therapists, and on the seemingly tardy adoption of family therapy approaches by those working with alcoholics. Steinglass attributed this indifference to culturally based prejudices and stereotypes held by many health professionals. These stereotypes portray the alcoholic as unmotivated, self-indulgent, and self-destructive, with the result that work with alcoholics is accorded relatively low status and priority. In addition, the fact that the problematic behavior, alcoholism, was typically manifested in a parent rather than a child made the transition to the adoption of systems-based family therapy more difficult, since family treatment approaches most often were utilized to deal with child-focused difficulties. At the same time, therapists dealing with alcoholics were steeped in individually oriented theories of alcoholism, and thus were skeptical of the basic premise of a systems orientation — that is, the notion that alcohol could play a stabilizing, adaptive, or

functional role when viewed from the broader perspective of the family system.

Early references in the literature to the family of alcoholics can generally be divided into two types. Preoccupation with the assumed psychopathology of the wives of alcoholics was a common theme in the early 1950s (Futterman, 1953; Whalen, 1953). Almost exclusively on the basis of clinical reports and case studies, a common personality type was postulated that depicted the wives of alcoholic men as neurotic, sexually repressed, and dependent. In addition, they were seen as having poorly controlled aggressive impulses and a latent hostility towards men (see McCrady, 1981). This psychopathology was presumed to precede marriage and to account for the choice of an alcoholic spouse. The other major theme portrayed the wife and family as victims of the alcoholic's abusive, self-destructive, and disruptive behavior and as having to adjust to the pressures and tension of living with an alcoholic (Bailey, 1963; Jackson, 1954). The effect of the drinking on families was emphasized, although the reciprocal notion that families affected both the course of the abusive drinking and its treatment went largely unnoticed.

The interactive relationship between family dynamics and abusive drinking was first explored in a number of studies or clinical trials that offered concurrent group therapy for the wives of alcoholics, in addition to the therapy offered to the alcoholic (Burton, 1962; Ewing et al., 1961; Gliedman, 1957; Pixley & Stiefel, 1963). In none of these studies was a systems-oriented treatment offered to either patients or their spouses. The treatment that was made available was typically psychodynamically oriented group therapy. The importance of these efforts, however, rests with their systematic inclusion of the wives of alcoholics in the treatment of alcoholics, along with a growing recognition of the importance of effecting change in marital and family dynamics in order to make possible the reduction or elimination of excessive drinking (Steinglass, 1976).

The adaptation and application of systems theory to work with alcoholics, both at a theoretical and clinical level, did not materialize until the late 1960s. At that time, several theoretical papers, as well as clinical trials, were reported. Ewing and Fox (1968) commented on the adaptive role of alcohol in establishing and maintaining a viable, homeostatic balance for certain couples and, more important, on the need to alter that balance if the drinking problem was to be effectively treated. Similar themes were developed by Steinglass and his colleagues (Steinglass et al., 1971a,b), who, in addition to elaborating on the adaptive role of alcohol within certain marital and family systems, suggested that for many families alcohol could be conceived of as a stabilizing rather than disruptive force. Berenson (1976) and Davis et al. (1974) also commented, from a systems perspective, on the adaptive consequences of drinking both in maintaining alcohol abuse and as factors to be considered by the family therapist in developing an intervention strategy. Finally, Bowen (1974), an influential family systems theoretician and clinician, extended a systems analysis and perspective to the phenomenon of alcoholism. He wrote:

How does alcoholism fit into systems concepts? From a systems viewpoint, alcoholism is one of the common human dysfunctions. As a dysfunction, it exists in the context of an imbalance in functioning in the total family system. From a

theoretical viewpoint, every important family member plays a part in the dysfunctioning of the dysfunctional member. The theory provides a way for conceptualizing the part that each member plays. From a systems theory viewpoint, the therapy is directed at helping the family to modify its patterns of functioning ... When it is possible to modify the family relationship system, the alcoholic dysfunction is alleviated (p. 117)

Thus, Bowen related a number of the basic tenets of systems theory to alcoholism: the interpersonal context within which it occurs; the need to change that context in order to alter the drinking behavior; the critical role that family members play both in the maintenance of the problem and in therapeutic efforts to effect change; and finally, the notion that alcoholism is a reflection of family or system dysfunctioning rather than of individual pathology.

Meeks and Kelly (1970) conducted one of the earliest studies of the effectiveness of systems-based family therapy with alcoholics. This study involved a sample of only five families and no control group. However, it did describe in some detail a systems-oriented treatment approach that involved conjoint therapy for the alcoholic and his family, following completion of an intensive program of individual and group therapy for the alcoholic. The goals of the family treatment, in addition to alleviating the drinking problem, were to improve family communication and to enhance problem-solving skills. Outcome measures included both alcohol-specific indices and assessment of change in family dynamics.

The work of Esser (1968, 1970) also involved conjoint therapy provided to the alcoholic and his family during the aftercare phase of treatment — that is, after the alcoholic was detoxified and stabilized by means of more traditional individually oriented therapy. Steinglass (1976) correctly described these works as "promising but unsubstantiated, enthusiastic but primarily impressionistic" since they fell far short of the well-controlled clinical trials necessary to establish the efficacy of a treatment approach.

Cadogan (1973) offered conjoint therapy to couples within a group context in addition to a traditional inpatient alcohol treatment program. This group was compared with a control group who received only the individually oriented inpatient treatment. The focus of the weekly group sessions was on improving family problem-solving patterns, facilitating the expression of feelings between the spouses, and enhancing their sensitivity to the interpersonal effects of their behavior. Follow-up indicated that the experimental group achieved substantially better outcomes in terms of their drinking problem.

Burton and Kaplan (1968) reported the results of a study in which marital-couples group counselling focusing on both drinking and interactional dynamics was offered. At follow-up, the results indicated that subjects receiving the marital-couples group therapy evaluated their therapy more positively and as more effective than did clients who received individual counselling. Gallant et al. (1970) also concluded on the basis of their study that marital-couples group therapy was the most effective treatment for married alcoholics. Couples involved in such treatment, following the completion of an inpatient phase of treatment for the alcoholic, showed improvement in relation to outcome measures dealing with both alcohol use and interactional dynamics.

Although all of these studies resulted in findings that suggested the possible efficacy of a myriad of family-based treatments for alcoholism, these findings remain subject to a number of different interpretations and any conclusions must be regarded as speculative. Such studies have, however, reinforced the enthusiasm of those already committed to the application of systems-based treatments to alcoholics and their families, and have also served as the springboard for further scientific and clinical work.

2.2 CONCLUSION

The application of systems concepts and techniques to the treatment of alcohol abuse is a relatively new development, dating back only to the 1960s. Although studies and clinical trials have generally confirmed the applicability and efficacy of such approaches, the evidence is by no means either definitive or conclusive. There have, in fact, been no well-designed, comprehensive studies. In light of the short history of the use of the approach in working with alcoholics, the field is, not surprisingly, still absorbed in developing treatment techniques and methods specifically for dealing with alcoholics, and in conducting and reporting small-scale clinical trials. However, the preliminary findings encourage the hope that a systems approach will clarify our understanding of alcoholism and lead to the development of efficacious interventions.

3. CHARACTERISTIC RESEARCH METHODS

Theory development within the systems approach has not, for the most part, been associated with rigorous research methodologies designed to operationalize key concepts. Rather, until recently systems research has typically consisted of clinical case studies in which interactions between family dynamics and a variety of clinical problems have been observed, documented, and discussed. In essence, these efforts have been largely directed towards assessing the fundamental assumption of systems approaches, that problematic behaviors are related to relatively stable interactional dynamics. Within the past few years, research in this field has become more systematic, focused, and sophisticated.

3.1 THE APPLICATION OF SYSTEMS CONCEPTS TO ALCOHOLISM

Systems-based treatment approaches emerged from a set of abstract ideas that were previously applied to problems in physics and biology. It was, therefore, necessary to translate such concepts into terms that were appropriate to the study of human relationships and interaction. Initial efforts at research were devoted to observing and studying families under stress and to applying systems concepts to the analysis of family behavior.

Much of the research during this critical initial period of applying systems theory to the phenomenon of alcoholism has been based on the strategy of systematically comparing interactional patterns of families or couples during periods of drinking and sobriety. These efforts often made use of experimental

drinking wards that permitted direct observation and analysis of interactional behavior and the role that alcohol played, rather than relying on more conventional self-report data (McCrary et al., 1979). This procedure provided an invaluable opportunity to test the conceptual framework of systems theory in a controlled research setting.

Within the field of alcoholism, the work of Steinglass and his colleagues stands out (Steinglass, 1979; Steinglass et al., 1977). This work involved the conjoint hospitalization of the alcoholic patient and his spouse in a "simulated apartment setting." Alcohol was made freely available during the first seven days of the period of hospitalization and couples were asked to engage in their typical drinking pattern during this period. The availability of alcohol provided an opportunity for the systematic observation of each couple's interactional behavior during periods of sobriety and intoxication. Observation techniques included videotape recording, use of one-way mirrors, and analysis of speech and communication patterns and nonverbal behavior. The results of this study led the authors to conclude that marital interactional behavior during periods of intoxication is more highly patterned and predictable than behavior observed during periods of sobriety. In addition, alcohol was seen as fulfilling an adaptive and functional role for certain couples. For some couples, affection, intimacy, and spontaneity were expressed only during periods when the alcoholic was drinking. By documenting such adaptive consequences of drinking from the perspective of the marital relationship, the authors confirmed a number of the central tenets of systems theory.

This line of research was further developed by Jacob and his colleagues (Jacob et al., 1981); who compared alcoholic and nonalcoholic families on a number of dimensions of family functioning, including communication, expression of affect, and problem-solving, in an experimental setting that included both drinking and nondrinking conditions. In total eight alcoholic and eight demographically matched nonalcoholic families participated in the study. Families were observed during both drinking and nondrinking conditions; analyses were conducted on videotaped segments of interaction involving various familial subsystems, and extensive interactional data were collected and analysed via reliable and theoretically based questionnaires.

Although clinicians and theoreticians had long been aware of the complex relationship between alcohol problems and familial or marital dynamics, such work represented an important breakthrough in systematically studying and identifying the interactional context within which alcohol problems occurred and were maintained. Alcoholism, in addition to its obvious destructive and debilitating effects on both individual and family functioning, was found to play an important adaptive and functional role in the context of marital and family functioning. For some couples and families, drinking was associated with the expression of warmth, affection, and caretaking behaviors; for others, drinking served to divert attention away from other potentially more threatening areas of conflict, such as the very viability of the marriage. In some instances, drinking served to legitimize and perpetuate changes in the roles and status of family members. Typically, the rituals and consequences of drinking provided an element of stability and predictability to interactional

dynamics, with subtle encouragement by family members for the continuation of drinking being evidenced. Such "adaptive" consequences were cited as evidence of the homeostatic role that drinking fulfilled in maintaining interactional systems (Davis et al., 1974).

3.2 ASCERTAINING THE UNIQUENESS OF FAMILIES WITH ALCOHOL-RELATED DIFFICULTIES

Having affirmed the relevance and usefulness of a systems-based perspective to the problem of alcoholism, researchers then turned their attention to exploring the possibility of identifying the unique systems-level characteristics or attributes of couples and families with alcohol-related difficulties. The operative question became: In what ways are alcoholic families, *as families*, different from normal families and from families manifesting other clinical difficulties? In attempting to illuminate this issue, Orford (1975), Gorad (1971), Wadsworth et al. (1975), and Jacob et al. (1981) have either speculated about or described characteristic interactional dynamics or attributes of family systems and subsystems with alcohol-related problems. Work in this area is, however, still in its early stages, with little consensus having emerged among either clinicians or researchers concerning the unique interactional qualities of alcoholic families or couples.

3.3 THE STUDY OF FAMILY PROCESS

An important focus of research over the course of the past few decades has been the study of family process (Wynne, 1983). The major objective of such efforts has been to generate a better understanding of the dynamics of family functioning and interactional behavior, in both normal families and in families presenting with a clinical problem. Such studies can be thought of as basic research, in that they are geared to identifying and elucidating key factors in the establishment and maintenance of stable interactional patterns within families and, secondarily, to identifying those dynamics most amenable and responsive to therapeutic interventions. The work of Riskin and Faunce (1972) and Epstein and his colleagues (1978) exemplifies such attempts to capture the essence of how families function, while Steinglass and his colleagues (1977), as indicated earlier, have done a considerable amount of pioneering work in the narrower field of studying process variables associated with alcoholic couples in both "wet" (drinking) and "dry" (abstinent) states. A corollary to this line of research has been the detailed and systematic examination of the process by which families change during therapy. Pinsoff (1981) describes the various methods utilized in such research on the process of family therapy, including self-report measures, instruments completed by family members and therapists, direct observation, and the application of coding systems (rating scales), by trained coders, to transcripts or tapes of therapy sessions.

3.4 THE ANALYSIS OF CASE MATERIAL

Much of the research and theory development that have been reported in the systems literature relied upon detailed reports of case material (Wynne, 1983). Until quite recently, such efforts were probably the most characteristic

research activity within this field. Family interviews, for instance, were observed or videotaped and then reviewed by clinical colleagues in order to develop a set of hypotheses concerning the current problem and a plan to intervene. This process, in turn, would lead to attempts to develop and operationalize innovative treatment techniques. Such techniques would then be implemented in work with a family or a small number of families and evaluated. Essentially, this has been the way in which the family therapy field has adopted many new treatment strategies and techniques, including the use of paradoxical injunctions (Palazzoli-Selvini et al., 1978). Wynne (1983) suggests that the current rapprochement between clinicians and researchers within the family field reflects a return to the "case study" approach to family therapy research, an approach that characterized research in this field in the 1950s.

3.5 GUIDELINES FOR TREATMENT OUTCOME RESEARCH

Perhaps the area of greatest progress in terms of research within the systems field has been in the specification of a set of criteria to ensure the adequacy and rigor of treatment outcome studies. In this regard, the contributions of Gurman and Kniskern (1978) have been particularly noteworthy. Their guidelines for evaluating the adequacy of systems-oriented treatment outcome studies include the following criteria (pp. 820-821):

1. Controlled assignment to treatment conditions.
2. Pre-postmeasurement (*sic*) of change.
3. No contamination of major independent variables.
4. Appropriate statistical analysis.
5. Follow-up.
6. Treatments equally valued.
7. Treatment carried out as described or expected.
8. Multiple change indices used.
9. Multiple vantage points used in assessing outcome.
10. Outcome not limited to change in the "identified patient."
11. Data on other concurrent treatment.
12. Equal treatment length in comparative studies.
13. Outcome assessment allows for both positive and negative change.
14. Therapist-investigator nonequivalence.

The presentation of this set of criteria for marital and family therapy research has been very significant. Although there is considerable overlap between these criteria and those put forward to evaluate other forms of psychotherapy, there are several items that are uniquely important and relevant to the field of outcome research in marital and family therapy. The criterion of ensuring that outcome is not defined solely in terms of change in the status of the "identified patient" is particularly noteworthy in this regard. Since systems therapies are premised on the interrelationship between problematic behaviors and ongoing marital or family dynamics, it is not surprising that emphasis is placed on measuring outcome at both the identified patient (individual) and marital/family (system) level. Gurman and Kniskern (1981) suggest that the identified patient, marital relationship, and total family system

all must be assessed in any marital/family therapy outcome study.

Along with the use of multidimensional criteria to evaluate outcome in family therapy, assessment of change from multiple perspectives is also required (Gurman & Kniskern, 1981). The unique perceptions and judgments of different family members are all essential components in developing a comprehensive systems-level evaluation of treatment outcome. Relying exclusively on the subjective perceptions of a family member, typically the identified patient, to evaluate family or subsystem functioning is clearly inadequate from a systems perspective.

Notwithstanding the fact that virtually all of the existing studies of the efficacy of systems-based treatment fall far short of these standards, the very existence of an explicit set of design and operational criteria for such research has had a discernible effect. Concerted efforts have been made to overcome the deficiencies in the standard research methodologies typically used in family treatment research, deficiencies that were made apparent by the general acceptance of these guidelines. For instance, family-based rating scales and standardized assessment procedures are being utilized more extensively to establish pre- and post-therapy scores, from which one can assess change (Wynne, 1983). Similarly, there is a much more concerted effort to describe precisely the treatments being given to families and to include provisions in the research design to ensure that the treatment delivered conforms to the treatment described in the protocol. Whereas behaviorally oriented researchers have long incorporated such provisions in their work, much of the research heretofore conducted with couples and families has consisted of the evaluation of the effectiveness of treatments that were not adequately described. For the most part, the treatments were simply referred to as "family therapy," "communication-interactionally oriented treatment," "systems-based treatment," etc. (see Slipp & Kressel, 1978). As a consequence of this lack of precision concerning the nature of the treatment being evaluated, virtually no studies within the family therapy field have incorporated measures to ensure therapist compliance with and conformity to the therapeutic regimen as prescribed in the research protocol.

Finally, the emergence of a technology and set of guidelines for conducting outcome research has provided a much-needed impetus to both clinicians and researchers. The antipathy that previously characterized relationships between family therapists and researchers is gradually being replaced by mutual respect and collaboration. The anticipated result of such trends is further sustained growth in the quality of outcome studies in which systems-based therapies are being evaluated.

4. BOUNDARIES OF THE THEORY

Systems theory has been applied to the study and treatment of an increasingly diverse range of clinical problems over the past few years. Although initially developed as an alternative approach to dealing with such intractable forms of psychopathology as schizophrenia (Bodin, 1981), systems

theories were subsequently applied to other difficulties, most of which were conceptualized as being essentially interactional as opposed to psychopathological in nature. For example, marital distress, child-rearing difficulties, family conflict, and child-centred problems in general typically were the kinds of problems that lent themselves to systems-based treatment approaches. The application of systems theory to the analysis of other clinical problems that were traditionally conceptualized in individually oriented psychodynamic terms, and that manifested themselves in a parent as opposed to a child, progressed more slowly. This traditional distinction accounts, in part, for the relatively late adaptation of systems concepts and theory to the field of alcoholism (Steinglass, 1976). More recently, however, alcoholism treatment personnel have demonstrated much greater interest in the approach. It has been shown to be readily adaptable to working with alcoholics and their families. In spite of these developments, there remain a number of limitations concerning the theory and its applicability to the phenomenon of alcoholism.

4.1 ETIOLOGY

Systems theory does not purport to be an etiologically oriented theory. In fact, the theory clearly does not lend itself to detailed, comprehensive formulations concerning the origins of problematic behaviors, the early manifestation of such behavior, or the manner in which the family's interactional behavior became transformed and rigidified to accommodate the behaviors. No doubt reflecting the unreliability of information obtained from different family members concerning the causes and historical development of current problematic behaviors, systems theory has emphasized factors associated with the maintenance and persistence of such behaviors rather than with their origin. It is, as such, a present- and future-oriented epistemology, and, in this regard, stands in sharp contrast with psychodynamic approaches, which typically involve theories of causality implicating early infantile experiences and hence demand retrospective analyses.

The failure to incorporate an etiological perspective represents a serious deficiency in a theory of human behavior. Clearly, a theoretical approach must account for three major domains of phenomena: the causes or course of development of specific behaviors, the maintenance or persistence of such behaviors, and the process of changing the behaviors. At present, systems theory offers very little in terms of dealing with the first of these three areas. Thus, it is relatively silent when it comes to addressing issues such as the type of family interactional dynamics that are associated with the emergence of alcohol-related problems rather than other problems. The theory also does not adequately take into account other important issues such as identifying the processes that determine which family member or members assume the role of identified patient. Also left unclear is the significance that should be attached to traumatic events in the family's developmental history, in attempting to understand the subsequent emergence of problems such as alcoholism.

4.2 SYSTEMS VARIABLES RELATED SPECIFICALLY TO ALCOHOLISM

The systems field, as is the case with other theoretical approaches, is still

attempting to develop a comprehensive set of hypotheses uniquely related to the phenomenon of alcoholism. Although a number of investigators and theoreticians are in the process of trying to identify and isolate the distinguishing interactional dynamics of families or couples in which alcoholism is a problem, there is the strong possibility that such efforts may be as frustrating as the earlier search for "the alcoholic personality." Until recently the notion that there is a unique constellation of personality variables associated with the alcoholic has been a predominant theme within psychodynamically oriented psychotherapy. At this time, there is general acceptance of the fact that no such unitary syndrome exists and that alcoholics, rather than constituting a unique personality type, are an extremely diverse and heterogeneous group (Pattison, 1966). (See Cox, Chapter 5 in this volume, for the current status of "personality theory.")

The discovery of unique systems-level markers of alcoholic families and couples may prove as elusive as the notion of the alcoholic personality. Orford (1975), for instance, strongly suggests that there may be nothing unique in the marriages of alcoholics, but rather such marriages tend to resemble other marriages that are characterized by stress, unhappiness, and crises. McCrady (1981) also tentatively suggests that alcoholic couples do not appear to be dramatically different from other distressed couples in their communication styles, problem-solving skills, and reliance on coercive controls, threats, and nagging. Rather, the abusive use of alcohol as a means of dealing with conflict, threats to stability, and the expression of affect may be the only way in which such families or couples are unique. In other words, the manner in which distress, dysfunction, or disequilibrium is expressed may be the only variable that differentiates alcoholic families from other distressed families. Such findings may preclude the development of primary prevention and early identification strategies by which families at risk of subsequently manifesting problems with alcohol could be identified. Similarly, the failure to discover unique systems-level characteristics of alcoholic families makes the task of clinical intervention with such families more complicated and varied. In the absence of a prototypical alcoholic family, a unique treatment plan based on a comprehensive family-based assessment is required for each family, rather than simple adoption of a standardized intervention strategy developed generically for "alcoholic families." The search for a common "family type" to account for the emergence of alcohol problems is motivated, in part, by this desire to enhance primary prevention and early identification efforts, as well as to make treatment more efficient and effective. Systems theory still has not resolved this issue of unique systems-level characteristics of alcoholic families, and no resolution appears imminent, at least in the short run.

4.3 SYSTEMS-LEVEL CHANGE VERSUS RESOLUTION OF ALCOHOLISM

Systems therapists are divided about the goal of clinical intervention. In families with a clearly defined presenting problem (e.g., alcoholism) expressed through the behavior of one family member, should the goal be first-order or

second-order change? Does systems theory imply that systems-level change is a necessary precondition for the amelioration of the presenting problem? Or does systems theory simply provide a broader conceptual basis for working specifically and immediately on the presenting problem, with the expectation that the family can be helped to change once the presenting problem abates? Structural therapies tend to subscribe to the former point of view, whereas strategic therapies tend to be much more oriented to directly addressing the presenting problem.

While there is widespread agreement that there is a strong, positive correlation between enhanced family functioning and improvement in the drinking problem (see Steinglass, 1979), systems theory has not clarified the nature of the relationship between these two sets of phenomena. Will marital interaction and satisfaction improve as a natural consequence of abstinence or non-problematic drinking? Or will alcohol-related problems diminish as a result of interventions aimed at helping the couple achieve a new level of functioning? In the absence of a resolution of this fundamental issue, systems therapists are increasingly being encouraged to focus on the drinking problems, albeit from an interactional perspective, during initial phases of treatment (McCrady, 1981; Zweben & Pearlman, 1983). Such an approach is consistent with the expectations and needs of families seeking treatment and is, therefore, more likely to be effective in engaging the family in the treatment process (Haley, 1976). Since, in most cases, problems with alcohol are what prompted the family to seek therapy, the initial focus of treatment should both reflect and be responsive to such concerns. It is recommended that as progress in terms of the drinking problem is achieved, the focus of treatment should then broaden to include general interactional dynamics during subsequent stages of treatment.

4.4 OTHER LIMITATIONS

Like other theories of psychotherapy, systems theory clearly does not encompass or adequately deal with all of the variables thought to be associated with alcoholism. As indicated, intrapsychic factors such as personality variables and psychopathology are not emphasized in a theoretical model that gives ascendancy to interactional, interpersonal dynamics. Similarly, demographic, societal, cultural, and economic factors are not directly incorporated into a systems perspective, although systems therapists are concerned with transactional systems that transcend the nuclear and even the extended family. Genetic aspects of alcoholism, stressing the possibility of a hereditary predisposition to alcoholism (Goodwin, 1976), are not addressed within a systems perspective. The pharmacological properties of alcohol as a drug, and their role and importance in the treatment of alcoholics, are beyond the scope of the model. Alcohol-related phenomena such as craving, loss of control, and tolerance are not directly covered by systems theory. Finally, theories concerning the mechanisms by which systems actually elicit and maintain behavioral repertoires are largely borrowed or adapted from social learning theories. The similarities between systems and social learning theories are discussed further later in this chapter (§ 7.2).

5. RESEARCH RESULTS AND NEEDS

In the Second Special Report to the United States Congress on Alcohol and Health, family therapy was described as the most notable advance in the field of psychotherapy for alcoholism (Keller, 1974). Although there are a number of studies that have offered tentative support for such a conclusion, the studies themselves have been so limited in their methodological rigor and so diverse in terms of the kinds of treatments included under the rubric of "family therapy," that both caution and conservatism are essential when interpreting the research findings on the efficacy of systems approaches to the treatment of alcoholism. More important, however, replication of existing studies and new studies incorporating features such as random assignment to treatment conditions, appropriate control conditions, larger sample sizes, improved pre- and post-treatment measures, and adequate follow-up are essential to establish the efficacy of this approach. Because of its methodological difficulties, the existing research offers, at best, only modest support to claims concerning its effectiveness (Miller & Hester, 1980).

5.1 THE OVERALL EFFECTIVENESS OF FAMILY THERAPY

A number of comprehensive and critical reviews of the effectiveness of marital and family therapy have recently appeared in the literature (Beck, 1975; Gurman, 1973; Gurman & Kniskern, 1978, 1981; Wells et al., 1972; Wells & Dezen, 1978). The general view emerging from these reviews is one of cautious optimism. Gurman and Kniskern (1978), for instance, report an overall improvement rate of approximately 65% for marital and family therapy, a figure that corresponds closely to the rate cited for individual therapy (Bergin, 1971). Unfortunately, however, the usefulness of such undifferentiated tests of the global efficacy of treatment approaches without regard for issues such as patient, therapist, and setting variables, and differing theoretical and clinical orientations, has been strongly questioned (Paquin, 1977; Paul, 1967).

These limitations notwithstanding, Gurman and Kniskern (1981) suggest that marital and family therapies are more effective than individually oriented psychotherapeutic approaches in dealing with marital or other problems that are interactional in nature. They further contend that marital and family therapy may also be more effective in dealing with some problems that are presented as being individualized or intrapsychic, rather than interpersonal. In their review of the literature, they specifically identify alcoholism as a phenomenon for which marital therapy and family therapy appear to be not only effective but preferred treatment approaches. The next section deals directly with the empirical evidence relating to this latter claim.

5.2 THE EFFICACY OF FAMILY THERAPY FOR ALCOHOLISM

Earlier in the chapter (§ 2.1), the developmental work in the area of applying systems concepts and treatment approaches in the field of alcoholism was reviewed. Much of this work took the form of clinical trials that reported outcomes for couples or families that had been engaged in exploratory forms of

systems-based treatments. A review of that material will not be repeated at this time. Rather, this section will feature an overview of the research findings and issues relating to the effectiveness of family approaches in the treatment of alcoholism.

It is worth noting at the outset that the involvement of the spouse in the assessment and treatment process has been associated with lower rates of attrition from outpatient alcoholism treatment settings (Gerard & Saenger, 1966; Gliedman et al., 1956). However, since social stability is predictive of successful treatment outcome and being married is an indicator of social stability, further research controlling for social stability is necessary to elucidate the precise impact of spouse involvement on reducing client-initiated premature termination of treatment. Similarly, the spouse's involvement in treatment may also be an important variable relating to the issue of treatment outcome, regardless of the nature, duration, and intensity of that treatment (Finlay, 1974; Gerard & Saenger, 1966; Smith, 1969). For instance, in a study conducted by Edwards and his colleagues (Edwards et al., 1977; Edwards & Orford, 1977), a "plain treatment" consisting of a single session of advice attended by the alcoholic and his spouse, along with extensive follow-up sessions also involving the spouse, was found to be as effective as the more traditional and extensive program of therapy that included a wide range of treatments that were made available to individual patients. Again, however, conclusions concerning the relationship between spouse involvement and treatment outcome remain speculative, since the existing research has not adequately dealt with the impact of other intervening variables.

Clearly, these findings are not in themselves directly relevant to the determination of the effectiveness of family treatment approaches. In some ways, such findings may in fact serve to further complicate that very issue. It appears that simply involving spouses and/or other family members may have significant implications for treatment outcome, regardless of the nature or orientation of the actual treatment that is subsequently provided. While this is hardly a surprising finding, it does suggest that future research will have to separate out the effects of involving family members from specific treatment effects in assessing outcome.

Treatment outcome studies in which various forms of family therapy served as the independent variable have revealed improvement rates, in terms of alcohol-related goals, ranging from about 45% to 80% (Miller & Hester, 1980). Again, while these improvement rates are encouraging, a number of factors must be taken into account before formulating conclusions. As has already been mentioned, virtually every study in this field is deficient in one or more crucial areas of design. The studies, for the most part, did not control for client characteristics such as social stability, nor did they use control groups or random assignment to the different treatment conditions. They typically relied on extremely small samples that made statistical analyses and inferences difficult. Many used therapist evaluations and patient self-reports to determine treatment effectiveness. In addition, the fact that the family treatment itself varied widely from study to study makes comparisons, generalizations, and overall conclusions very difficult. For instance, "family treatment" in the

outcome studies reported in the alcohol field has referred to: treatment offered in the recovery phase after a period of individualized inpatient therapy (Esser, 1968; Meeks & Kelly, 1970); concurrent group therapy for alcoholics and their spouses (Ewing et al., 1961); multiple-couple and multiple-family group therapy approaches (Cadogan, 1973; Gallant et al., 1970); joint admission of the nonalcoholic spouse along with the alcoholic to an inpatient hospital treatment facility (Corder et al., 1972; McCrady et al., 1979); and behavioral family therapy (Hedberg & Campbell, 1974). Other studies involving family therapy approaches used such draconian contingencies as removing children from the home until the drinking problem was resolved (Amir & Elder, 1979). All these variations render findings difficult to interpret in terms of ascertaining the specific family treatment effect.

Under these circumstances, conclusions concerning the effectiveness of family therapy must be tentative. It is worth noting, however, that all of the studies that have incorporated adequate research designs generated findings that confirmed the efficacy of couples therapy in increasing the likelihood of improvement in drinking behavior (Cadogan, 1973; Corder et al., 1972; Hedberg & Campbell, 1974; McCrady et al., 1979). These findings strengthened the enthusiastic support for family therapy approaches emanating from uncontrolled or inadequately controlled clinical trials.

5.3 RESEARCH ON SYSTEMS THEORY APPROACHES IN THE FIELD OF ALCOHOLISM

The marital and family therapy field is extremely broad and diversified. Family treatment based on systems theory represents only a subset of this larger field. In this section, only those studies clearly falling within the purview of systems theory approaches will be reviewed.

There have, in fact, been very few reports in the alcoholism literature of treatment outcome studies that were clearly identified as being applications of systems theory. Although most studies made reference to the family as a system, and incorporated elements of systems theory in their treatment model, the treatment that was evaluated was, for the most part, predominantly psychodynamic. Typically, the treatment approach was modified to allow for the participation of the spouse or family, where such participation was seen as a form of clinical leverage in effecting change in the alcoholic (see Gliedman et al., 1956). More recently, there have been a number of studies in which families have been involved in a behaviorally oriented treatment regimen (Hedberg & Campbell, 1974). Although such studies have consistently reported positive outcomes, they do not provide a definitive answer to the issue of the usefulness of systems theory in working with alcoholics.

Meeks and Kelly (1970) reported positive outcomes in terms of both improved family interaction and also drinking behavior on the part of the identified patients. This study, which clearly was exploratory in nature, was severely limited by such factors as its small sample size (five families) and lack of control group. The study did, however, elaborate a treatment model that was based on systems theory. Similarly, 6-month follow-up data reported by Steinglass (1979) on nine couples who participated in a treatment program that

involved conjoint hospitalization of spouses revealed modest improvement in drinking behavior for five of the nine subjects, although measures of marital functioning showed little improvement other than consistently positive changes in communication. This improvement in communication was counterbalanced by perceptions of increased behavioral difficulties and decreased satisfaction in other areas of marital life. Steinglass, while acknowledging the speculative nature of the findings, suggested that the study reaffirmed the validity of a conceptualization of alcoholism as a family-level process and emphasized the relative importance of changes in interactional behavior as a specific goal of treatment.

A study currently in progress probably represents the most elaborate and ambitious study of the effectiveness of systems-based treatment for alcoholics (Pearlman & Zweben, 1981; Zweben & Pearlman, 1983; Zweben et al., in press). In this study, alcoholics and their spouses are randomly assigned either to an experimental treatment consisting of short-term conjoint outpatient therapy or to a control condition in which each couple is seen for a single treatment session. The control condition was adapted from the earlier work of Edwards and his colleagues (Edwards & Orford, 1977; Edwards et al., 1977). The conjoint therapy is offered as the primary treatment immediately after the assessment stage, as opposed to being made available in the aftercare or recovery phase. A large sample (60 couples in each treatment condition), extensive pre- and post-treatment measures focusing on both marital dynamics and drinking behavior, independent follow-up over a 18-month period, and the use of both the patient and spouse as sources of data during all data collection contacts are important features of the study. In addition, provisions for the training, supervision, and monitoring of therapists are designed to ensure that the therapy is offered consistently and in accordance with the research protocol. All sessions, for instance, are audiotaped and then subjected to a random review to ensure that the therapist is maintaining an interactional focus and that standards of skill and competence in the use of the model are met. It is anticipated that this study, when completed, will constitute perhaps the only controlled clinical trial of the application of systems-based marital or family treatment in the alcoholism field. As such it should contribute significantly to the determination of the efficacy of this approach.

5.4 RESEARCH NEEDS

More and better research is required in the systems field generally and in the alcoholism treatment field more specifically. There is an increasing awareness of, and commitment to, systems-based therapy approaches among alcohol treatment personnel, and this development clearly requires an equal commitment to the systematic evaluation of the efficacy of these approaches. Guidelines for conducting treatment outcome studies within the systems field, although requiring elaboration and refinement in some important dimensions, have been developed and adopted. The studies of structural family therapy with drug addicts are now considered to be among the best-controlled outcome studies in the field of family therapy (Gurman & Kniskern, 1981). Similar progress can realistically be anticipated in the closely related area of the treatment of alcoholism.

Research in the systems field has been hampered by the relative absence of clinical psychology, the discipline typically most actively involved in psychotherapy research (Stanton, 1975). Theoreticians and clinicians with an interest in systems-based family therapy have, for the most part, been drawn from the ranks of psychiatry and social work. Although these two disciplines are developing greater sophistication in the area of research, the cause of research would certainly be enhanced if there were more active participation and involvement on the part of clinical psychologists in the general area of systems theory and therapy.

Perhaps no area commands higher priority in research than an examination of the complex relationship between changes in the identified patient's presenting problem (e.g., alcoholism), on the one hand, and changes in family functioning, on the other. Clearly, if systems theory is to be accepted as a comprehensive theory of human behavior and behavioral change, it must directly address the relationships between systems-level changes and changes in individual behaviors. In this regard, therapists dealing with distressed families or marriages in which alcoholism is present should be able to expect that the treatment model they adopt will provide them with more specific guidance concerning treatment strategies and priorities. As McCrady (1981) points out, the nature of the relationship between individual and systems-level changes is ultimately an empirical question.

Although the central concepts and constructs of the systems model are discussed widely in the family therapy literature, very little progress has been made in terms of operationally defining these concepts and developing valid measures of them. Gurman and Kniskern (1978) suggest that this failure to develop useful and valid measures of the central theoretical constructs of systems theory represents one of the most serious shortcomings in the field. They cite "enmeshment," "collusion," and "triangulation" as examples of constructs that, though widely accepted within the systems field, have not been adequately operationalized. Galligan (1982) echoes this same concern about the failure of systems theorists and researchers to translate abstract concepts and terms into functional descriptors of family dynamics.

It is hardly surprising in light of this lack of progress in terms of operationalizing key concepts that systems theory has generated very few valid instruments to assess and measure dimensions of family functioning. Unlike the situation within the realm of individual psychotherapy, there is no "core battery" of standardized measures of family dynamics or functioning (Gurman & Kniskern, 1978). Not only is there inadequate instrumentation, there is no consensus as to which dimensions of interactional behavior are most salient, relevant, and meaningful to the process of helping families to change. Systems therapies lag well behind behavioral approaches in terms of transforming theoretical constructs into quantifiable, measurable entities that lend themselves to scientific study and objective assessment.

6. PRACTICAL IMPLICATIONS

The implications of the application of systems ideas, theory, and treatment to the field of alcoholism are numerous and diverse. Although there continues to be considerable enthusiasm among those clinicians who have used and reported on the results of systems-based treatment approaches, their use has not become widespread in working with alcoholics. In part, this reflects the fact that systems-based marital and family therapies are relatively new and, in many ways, a direct challenge to more traditional conceptions of psychopathology, psychotherapy, and alcoholism. More important, however, there remain a number of unanswered questions concerning the use of such treatment approaches that must be effectively dealt with if systems therapies are to become more widely utilized.

6.1 THE INVOLVEMENT OF FAMILY MEMBERS IN THE TREATMENT OF ALCOHOLISM

It has been noted that the direct involvement of family members in the treatment of alcoholics seems to be associated with reduced rates of attrition from treatment (Gerard & Saenger, 1966; Gliedman et al., 1956). This finding is particularly significant in light of the distressingly high rates of attrition, especially in outpatient programs (Gerard & Saenger, 1966). Similarly, the relationship between alcohol problems and their resolution, on the one hand, and ongoing marital and family dynamics, on the other, has been observed by clinicians and theorists representing behavioral (Hedberg & Campbell, 1974; McCrady et al., 1979), systems (Steinglass, 1975), and other perspectives.

Systems theory provides a coherent rationale and theoretical framework to account for these important findings. In addition, it offers the foundation or framework upon which to develop a comprehensive treatment approach based on the direct involvement of family members in the assessment, treatment, aftercare, and follow-up stages of intervention with the alcoholic. Assessing marital and family dynamics and their relationship to the drinking problem, collecting data from the spouse and other family members, and enlisting the collaboration of family members in effecting change in drinking patterns are strategies that have been adopted with increasing frequency by clinicians and researchers in the alcoholism field. Such trends indicate an increase in the role that family members will play in the assessment and treatment of alcoholism, and in the evaluation of the effectiveness of that treatment. Systems theory clearly did not provide the initial impetus for these developments; nonetheless, it is in the forefront of theoretical approaches that focus on the family and its crucial role in redressing the problem of alcoholism. If this trend towards further involvement of the families of alcoholics in the treatment process continues, systems theory may become a more appealing conceptual model to program planners, clinicians, and researchers.

6.2 TEACHING AND TRAINING CONSIDERATIONS

Gurman and Kniskern (1981) observed that most marital and family therapy is being practised, and even being taught, by relatively inexperienced

clinicians. This observation on the general field of marital and family therapy is perhaps even more true of the more specialized field of systems-based treatment interventions. Indeed, one might further suggest that much of the research on systems treatment is being designed and conducted by researchers with relatively limited backgrounds in systems theory and practice. The need for a cadre of better-trained and more experienced clinicians, teachers, and researchers is both obvious and pressing.

To a significant degree, the current predicament of relatively poorly trained and inexperienced professionals is an inevitable result of the way in which systems theory is taught. Although many graduate faculties of social work have incorporated systems theory and systems treatment approaches into their curriculum, the same cannot be said for the disciplines of psychiatry, medicine, nursing, or psychology. As a consequence, relatively few therapists are thoroughly familiarized with systems treatment modalities during the course of their academic clinical training. Even within social work, courses in systems approaches are elective rather than integrated into the core clinical curriculum. Thus, many therapists become "systems" therapists by gravitating to clinical settings in which systems approaches are already entrenched; by participating in post-graduate training, conferences, or seminars that typically provide intensive but relatively brief training experiences; or, most typically, by developing sufficient familiarity and comfort with the basic elements of systems treatment so as to incorporate occasional marital or family interviews into their clinical practice.

The systems field has relied far too much on a few charismatic individuals who have exercised a profound and, at times, constraining influence on the training of professionals (see Beels & Ferber, 1969). Clearly, systems theory has matured to the point where more comprehensive, formal, and systematic provisions for the training of clinicians should become the norm rather than the exception. To this end, it is worth noting that a number of very useful guidelines for the training and evaluation of therapists have begun to appear in the systems literature (Cleghorn & Levin, 1973; Tomm & Wright, 1979), as well as several attempts to determine the extent to which training programs are successful in transmitting the skills they purport to teach (Breunlin et al., 1983; Flomenhaft & Carter, 1977; Tomm & Leahy, 1980). These are, indeed, most timely and encouraging developments.

6.3 THE SPECIFICATION OF TREATMENT

For the most part, systems theory has not been translated into clearly defined, explicit, and discrete interventions. Whereas structural family therapists and those utilizing paradoxical therapies have made some progress in this regard, systems therapies, in general, still remain relatively poorly specified. A notable exception is the work of Sluzki (1978), who has attempted to identify, specify, and illustrate a number of systems interventions to be used in marital therapy. Nevertheless, the lack of a well-defined treatment technology has proven to be a problem for both clinicians and researchers. Slipp and Kressel (1978), for instance, acknowledged the need for greater precision in defining the treatment variable in most family therapy outcome studies. They noted

that most studies, including their own, were limited by the absence of any valid assurance that the treatment being evaluated was delivered in a consistent fashion and in conformity with the research requirements. Indeed, there are relatively few marital and family therapy manuals that provide clinicians with a formal elaboration of actual interventions (Gurman & Kniskern, 1978).

There are still relatively few comprehensive descriptions of systems-based treatment approaches adapted specifically to the phenomenon of alcoholism, although the work of Berenson (1976, 1979), Kaufman and Pattison (1981), Nace et al. (1982), and Anderson and Henderson (1983) represents some progress in this respect. Systems therapists are still, however, at a distinct disadvantage when compared, for instance, with behavioral therapists in terms of having ready access to a set of well-defined, succinctly operationalized treatment interventions.

6.4 THE INTERVENTION "SYSTEM" IN SYSTEMS THERAPIES

Although systems theory and systems-based clinical interventions represent a rather dramatic broadening of the perspective within which behavioral problems are understood and ultimately dealt with, systems-based clinical interventions have themselves been criticized for being too preoccupied with family-level dynamics at the expense of extrafamilial issues such as work, leisure, and peer relationships. Such criticisms reflect the manner in which clinicians have applied systems concepts in their work rather than deficiencies in the theory itself. It is ironic indeed that many "systems" therapists focus exclusively on the family or even on subsystems within the family, while ignoring the larger context within which both the individual and family unit interact. For instance, systems therapists working with alcoholics have been reproved by their own colleagues for being preoccupied with the marital subsystem and not involving the total family system in therapy (Kaufman & Pattison, 1981). Similar concerns about the need to maintain a more "ecological" rather than the more common familial perspective in clinical work have been a common theme in the systems literature (see Attnave, 1980; Auerwald, 1968). It is important to stress, however, that this issue reflects a problem in terms of how systems theory has been applied in clinical practice rather than a limitation of the model or theory itself.

6.5 SYSTEMS THERAPY: TREATMENT OF CHOICE?

In the absence of controlled studies comparing systems-based treatment models to other clinical interventions, it is doubtful that even the most fervent advocate of systems treatment would seriously argue that it is the preferred treatment in all situations, even when dealing with a particular field of therapy such as alcoholism. There is also the problem of determining which systems treatment is to be adopted even in those situations where a systems approach is felt to be most appropriate. Is structural or strategic therapy, or some combination of these two, the preferred treatment?

In the field of alcoholism, these questions are extremely important. Where an alcoholic is isolated and not part of an intact family unit or peer group, or where the family is unwilling or unable to participate in treatment, systems therapy may simply be inappropriate. More important, however, even

when the family is willing to become involved in treatment, that factor may not, in itself, justify proceeding with systems-based family therapy. More knowledge is required to ascertain which families can best be treated through family or marital therapy and under what set of circumstances. For instance, is conjoint marital or family therapy more effective when alcoholism emerges as a problem subsequent to the marriage? Is systems therapy most applicable to situations in which marital or family distress has been specifically identified by a family member? What kind of treatment support or logistical arrangements are necessary to ensure that systems-based interventions can be offered in an effective manner to an alcohol-abusing population?

These are fundamental questions that ultimately must be answered. The alcoholism treatment field has unfortunately been characterized by attempts to identify single treatment models or approaches to be applied to all alcoholics. There is today a greater acceptance of the fact that a number of different treatment approaches and models will be required to effectively respond to the varieties of alcohol dependence (Jacobson, 1976; Pattison et al., 1977). The role of systems treatments within this repertoire, and the indications and contraindications for such treatment, have yet to be determined.

7. RELATION TO OTHER THEORIES

In this section of the chapter, the relationship of systems theory to three other approaches to the treatment of alcoholism will be discussed: the "disease theory," behavioral approaches, and psychodynamic theories.

7.1 "DISEASE THEORY" OF ALCOHOLISM

A systems perspective on alcoholism has very little in common with the "disease theory." Many of the more important aspects of the "disease theory" are simply not dealt with by systems theory. Thus, for instance, notions such as craving and loss of control, two of the central constructs of the disease model, are virtually unmentioned in the systems literature. Clearly, systems approaches focus on interactional dynamics rather than on subjective or physiological processes in terms of explaining how drinking is elicited and maintained. The notion of alcoholism being a progressive, irreversible process in which certain individuals manifest a peculiar susceptibility to physiological reactions after ingesting alcohol is not central to a systems approach.

Controlled or non-problematic drinking as a treatment goal is clearly incompatible with the "disease theory" of alcoholism, since the theory is predicated on the notion that alcoholics, as part of their unique reactions to alcohol, exhibit loss of control in terms of their drinking behavior even after a modest intake of alcohol. Initially, the systems literature espoused an essentially similar view, suggesting that abstinence was the only realistic and appropriate drinking goal (Berenson, 1976). This adherence to treatment goals defined in terms of abstinence appears, in retrospect, to be more a reflection of the more generally pervasive ideas about alcoholism at that time, rather than an essential feature of a systems perspective. For instance, Zweben and Pearlman

(1983) describe a research project designed to determine the efficacy of a conjoint systems-based therapy for alcoholics in which individual drinking goals are defined for each client in terms of either abstinence or non-problematic drinking. The issue within the systems model is entirely a practical and empirical one, with drinking goals being determined on a case-by-case basis depending upon factors such as the extent and duration of drinking, the preferences of the client, spouse, and other family members, evidence of alcohol-related physiological damage, and prior failure in treatment oriented to non-problematic drinking.

7.2 BEHAVIORAL THEORIES

Behavioral theories and systems theory are closely related in some important respects. Both theories conceptualize alcoholism as a behavioral pattern that is elicited and maintained by current forces, in contrast with psychodynamic formulations that typically conceptualize alcoholism as a manifestation or symptom of long-standing intrapsychic difficulties. From a behavioral perspective, alcoholism is related to the interplay of antecedent cues, consequences, and cognitive processes. From a systems viewpoint, alcoholism is related to a particular subset of environmental cues and consequences, namely marital, family, or larger systemic dynamics. However, while behavioral approaches maintain a focus on the individual in the development of a functional analysis of the problematic behavior, systems theory redefines the problem into an interpersonal context in which the individual alcoholic becomes the "identified patient" and the use of alcohol is seen as purposeful, adaptive, and meaningful in terms of ongoing interactional dynamics. Alcoholism thus becomes a family systems problem, with primary emphasis being placed on effecting systems-level changes to promote, enhance, or maintain changes in abusive drinking.

In spite of this distinction, there are important similarities between behavioral and systems approaches, at a practical or clinical level. These similarities include: an orientation to the present and future with little emphasis on historical material; a shared emphasis on the behavior of drinking rather than any assumed underlying pathology; a problem-oriented treatment approach that permits short-term therapies; an active, direct, and forceful therapist style; and the use of a number of the same treatment techniques (e.g., role playing, behavioral rehearsal, modelling, problem-solving training, communication training, and teaching family members positive reinforcement techniques).

7.3 PSYCHODYNAMIC THEORIES

Psychodynamic theories of alcoholism postulate the existence of underlying intrapsychic difficulties as the source of problems such as alcoholism. Thus alcoholism is conceptualized as a symptom of more basic emotional and psychological problems. Hence, treatment is geared towards alleviating the underlying psychopathology. By focusing on the presenting problem, the therapist would simply eradicate the symptom. In such instances, the failure of the therapist to deal effectively with the source of the difficulties would result in the emergence of new problems or difficulties (symptom substitution).

Thus, for example, providing disulfiram to an alcoholic without attending to the supposed intrapsychic causes of his drinking (e.g., anxiety, depression) will, according to this perspective, simply give rise to new modes of expressing the underlying psychopathology (e.g., sleep disturbance, agitation, inability to concentrate). Although there is very little empirical support for the notion of symptom substitution, it continues to be a fundamental premise of psychodynamic theory and clinical practice (Bandura, 1967).

At times, systems theorists also refer to alcoholism as a symptom, not of psychopathology within the alcoholic, but rather of dysfunctional interactional dynamics within the family or marital dyad. Systems theorists have alluded to the futility of focusing directly on drinking problems without effecting systems-level changes to facilitate and accommodate the alcoholic's relinquishing excessive drinking (Steinglass, 1979). Unlike the situation with psychodynamic theory, however, there appears to be a trend within systems approaches to focus more directly on the problems that prompted the individual or family to seek treatment in the first place (Haley, 1976). There is also a concomitant acknowledgment that changes in drinking behavior must precede and set the stage for changes in systemic functioning within the family. In this regard, it is interesting to note that the term "symptom" is being used less frequently in the systems literature. Instead, problems such as alcoholism are described as being "dynamically related to family functioning" (Kaufman & Pattison, 1981) or as fulfilling a systems-maintaining or homeostatic role for the family (Berenson, 1976; Pearlman & Zweben, 1981).

Although there is very little overlap between psychodynamic and systems approaches at the level of theory, many family and marital therapies have combined elements of both theories into a clinical modality that has been referred to as integrative or psychodynamically oriented family therapy (Pinsof, 1983; Stein, 1973). The evolution of this clinical approach, combining distinctive elements of both psychodynamic and systems theory, reflects the pragmatic eclecticism of many clinicians who attend to both intrapsychic and interpersonal forces when working with families or marital dyads. They suggest that ignoring either intrapersonal or interactional dynamics is arbitrary, constraining, and clinically futile. Instead of a "pure" systems approach, integrative approaches combine a basic systems orientation with a psychodynamic perspective that enables the therapist to focus on intrapsychic factors when such an emphasis is deemed to be appropriate or necessary.

For many clinicians working with alcoholics, the practical appeal of such an eclectic or integrated approach is very real and far outweighs the theoretical confusion that may result from combining two radically different orientations. Alcoholism is itself a problem that has traditionally been conceptualized in individualized psychodynamically oriented terms. In addition, many, if not most, of the "systems therapists" working with alcoholics have had far more exposure to and training in psychodynamic theory than in systems theory. The desire to integrate elements of both orientations when confronted with difficult and trying clinical cases is both natural and inevitable. An eclectic approach such as integrative family therapy may, indeed, be more flexible and responsive to differences among clients than a more dogmatic, though

theoretically consistent, adherence to a single theoretical and clinical perspective.

Notwithstanding the attractiveness and utility of integrative approaches, more research is needed to fully explore the promise and prospects of systems theory, to flesh out its value and limits, to describe the mechanisms by which systems operate, and to compare the effectiveness of systems therapies with other approaches.

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9. THE AVAILABILITY THEORY OF ALCOHOL-RELATED PROBLEMS

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I. PRINCIPAL ELEMENTS AND DEFINITIONS

The availability theory of alcohol-related problems asserts that the greater the availability of alcohol in a society, the greater the prevalence and severity of alcohol-related problems in that society. The availability theory is a relatively simple set of interrelated propositions describing how sets of variables are linked to one another. Before these specific hypotheses are presented, some key definitions will be introduced and the boundaries of the theory discussed.

The term "alcohol control measures" is used to refer to all government measures that affect the availability of alcoholic beverages. Thus, alcohol control measures include control laws at all levels of government, and include general economic regulations that affect all commodities in the market, not just those measures that treat alcoholic beverages as a special commodity. Alcohol "availability" refers to both physical accessibility (e.g., number of outlets and purchase restrictions) and economic accessibility (e.g., price and affordability).

A second and even more important point of clarification concerns the dependent variable in hypotheses derived from the availability theory. The major dependent variable is the general notion of *alcohol-related problems* rather than *alcoholism* per se. This notion is meant to include both health and social problems. Alcohol availability not only influences problems associated with clinical alcoholism such as cirrhosis and delirium tremens, but also a wide

variety of other problems, including alcohol-related violence, drinking and driving, industrial absenteeism, and low productivity.

The availability theory involves not only alcohol controls and alcohol-related problems, but also intervening mechanisms that require definition. The mean level of consumption in a society hardly requires clarification, but the terms "moderate" and "heavy" drinking are more controversial. For the purpose of this discussion, the term "heavy drinking" will refer to levels of consumption equal to or greater than minimal levels reported by persons who appear in alcoholism treatment settings. This term has been operationalized as the consumption of at least 15 cL of ethanol per day (about nine standard drinks) on the basis of the work of de Lint and Schmidt (1968). The term "moderate drinking" will refer to any level of drinking below this; thus, the term is used for a wide range of drinking behavior, ranging from very light, occasional use to levels of consumption approaching those of clinical alcoholics. In other circumstances, it would be useful to further articulate this category, but for the present purpose it is sufficient to merely distinguish "moderate" and "heavy" drinking.

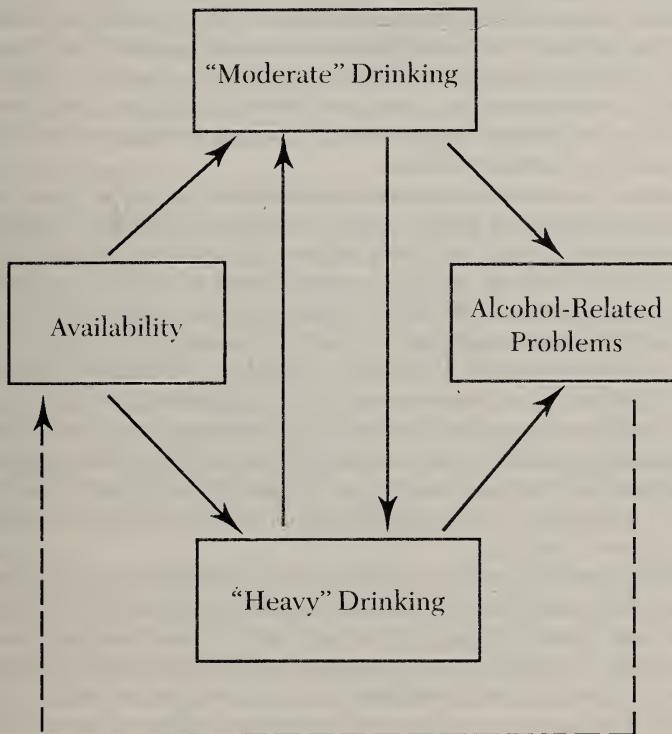
Alcohol availability and alcohol problems are theoretically linked in several ways. The first is the developmental sequence whereby increased availability is related to increased consumption by moderate or social drinkers, who then influence heavy drinkers to consume more. Heavier drinking is in turn related to increased incidence of acute and chronic health and social problems. This process involves three interconnected hypotheses:

1. Alcohol availability is positively related to mean levels of consumption:
As we shall see, controls on the availability of alcohol, such as age limitations, restrictions on the types of outlets, density of outlets, hours of operation, marketing and promotion of alcoholic beverages, and price of alcohol — all tend to be related to mean levels of consumption over time.
2. Mean consumption is related to levels of heavy drinking: The mean level of alcohol consumption in any society is closely related to the number of persons consuming at levels deemed to be high risk.
3. Heavy drinking is associated with adverse health and social consequences: This aspect of the theory is probably the least controversial. The probability and severity of adverse health effects of alcohol are strongly related to level of intake. The dose-response relationship is most evident with respect to cirrhosis, but adverse effects of high intake of alcohol have also been found for many other disorders including delirium tremens, impaired brain function, cancer of the esophagus and digestive tract, chronic calcifying pancreatitis, and congenital defects in the fetus among pregnant women. High alcohol use is also strongly associated with social consequences such as drunk-driving problems, industrial absenteeism, and poor productivity.

Thus, the most frequently cited argument for controls on alcohol availability as a preventive measure is based on these reasonably well substantiated hypotheses. However, as seen in Figure 1, this represents only one of several theoretically possible connections between availability and alcohol-related problems.

FIGURE 1

The relationship between alcohol availability and alcohol-related problems



Linkages Between Availability and Adverse Consequences

- (1) Availability → Moderate drinking → Heavy drinking → Alcohol-related problems
- (2) Availability → Moderate drinking → Alcohol-related problems
- (3) Availability → Heavy drinking → Alcohol-related problems
- (4) Availability → Heavy drinking → Moderate drinking → Alcohol-related problems

Moderate or social drinking not only influences heavy consumption, but also has a direct impact on the incidence of alcohol problems. The relationship of intake to risk is a continuous function for practically every alcohol-related problem. There is no threshold at which the individual suddenly acquires a risk of incurring a particular adverse consequence of drinking. In a review of alcohol-related morbidity and mortality studies, Schmidt and Popham (1975-76) concluded that the lowest level of consumption at which there is a significantly higher risk cannot be determined. Even moderate drinking can increase the probability of being involved in a traffic accident (Selzer, 1975). Similarly, relatively moderate drinking over a prolonged period of time significantly increases one's risk of developing cirrhosis (Péquignot et al., 1974). Thus, even when in moderation, drinking is associated with alcohol-related health and social problems.

Another connection between availability and alcohol-related problems derives from the finding that changes in access to alcoholic beverages will influence the consumption not only of moderate social drinkers but of heavy drinkers as well. Counter to what proponents of a "disease concept" might have expected, reduced availability has resulted in reduced drinking on the part of persons who consume alcohol at levels associated with clinical alcoholism. In Finland (Mäkelä, 1974) and in Sweden (*Alkohol Konflikten*, 1965), strikes by alcohol monopoly workers closed off-premise outlets for alcoholic beverages while on-premise sites such as restaurants and taverns remained open. During these strikes heavy drinkers reduced their consumption considerably, in roughly the same manner as did moderate drinkers. These studies indicate that changes in the availability of alcohol can have a direct and swift impact on heavy drinking.

Finally, there is another manner in which availability may influence the incidence of alcohol-related problems: increased availability of alcohol may influence heavy drinking, which in turn influences the consumption of moderate drinkers in an upward direction, which is then associated with increases in indices of alcohol-related problems. Small-group experiments on alcohol consumption have consistently found a strong modelling influence of drinking peers (see, e.g., DeRicco, 1978). Further, heavy drinkers may have a particularly marked influence on the consumption rate of their drinking companions. Bruun's classic work *Drinking Behavior in Small Groups* (1959) described drinking norms whereby a group member was permitted to drink more, but not less, than other members of the group. If such norms apply elsewhere, it would indicate that just as moderate drinkers can influence the consumption of heavy drinkers in an upward direction, so too heavy drinking can spur on the development of alcohol problems among social drinkers.

In sum, the availability of alcohol has a mutually reinforcing impact on drinking among *both* moderate and heavy drinkers, and consequently availability influences the incidence of alcohol problems. When formulated in this way, the crucial intervening mechanism by which availability is connected to problems is drinking in general, not just heavy drinking.

2. BOUNDARIES OF THE THEORY

As described above, the availability theory in its most general formulation holds that the greater the availability of alcohol in a society, the greater the prevalence and severity of alcohol-related problems. This does not in any way deny the importance of the particular cultural context of alcohol use: in the aforementioned International Study of Alcohol Control Experiences (Mäkelä et al., 1981) it was found that both the patterns of drinking and societal reactions to drinking problems differed considerably from one society to another. Indeed, for particular alcohol-related problems there are instances where a country appears to enjoy easy access to alcohol and yet has low rates of adverse consequences.

In this regard France is occasionally presented as a model of moderation because alcohol is readily available and the French enjoy the highest rate of consumption of any major country in the world, and yet the French have very little public drunkenness. Such examples, however, are somewhat superficial. The French may have relatively little public drunkenness, owing to the prevalent pattern of drinking gradually throughout the day, but they do experience very severe rates of other alcohol-related problems, particularly cirrhosis. Thus, even though it may appear to the casual observer that the French drink a great deal without adverse social consequences, a closer examination shows that they pay a high clinical price for their permissiveness. This example underscores the need to examine the full array of adverse health and social problems associated with alcohol use, rather than any one particular consequence.

Furthermore, it should be noted that the availability theory does not hold that access to alcoholic beverages is the sole or even the primary determinant of alcohol-related problems. An individual's risk of experiencing adverse effects from drinking can be broken down into two elements: exposure and vulnerability (WHO, 1980). For particular alcohol-related problems, there are clearly differences in vulnerability according to age, sex, occupation, and even heredity, but such factors are generally beyond the influence of public policy. What can be affected is the degree to which individuals are exposed to alcohol, which, according to the availability theory, does have a demonstrable impact on the incidence of alcohol-related problems.

It should also be noted that the theory only concerns the impact of availability on the incidence and prevalence of alcohol problems. The causal direction of the relationship between alcohol availability and alcohol-related problems is not simply one-way. Just as availability can influence alcohol-related problems, so too the incidence of adverse consequences can affect alcohol control measures. This observation was made in the report of the International Study of Alcohol Control Experiences:

It became clear that controls are not rigid systems of legal regulations which are easily manipulated. Rather, they are elaborate networks of cultural, economic and political structures which are both a response to and a determinant of the magnitude of alcohol-related problems. (Single et al., 1981, p.2)

A complete model explaining the interrelationship between availability and alcohol-related problems would be reiterative. It would necessarily include theoretical connections leading from the incidence of alcohol-related problems to the control system via the economic, cultural, and political system. To fully describe the interrelationship would therefore require what Zetterberg (1965) has termed a "grand theory" involving all the major aspects of society and social change. The availability theory is not such a theory. It is focused exclusively on the influence of availability on problems and does not attempt to deal with the reiterative effects of the magnitude of alcohol-related problems on the alcohol control system.

3. HISTORICAL DEVELOPMENT OF THE AVAILABILITY THEORY

Over the past two decades there has been increasing interest in the use of controls over availability as a means to prevent alcoholism and related health and social problems. In the recent past, a number of international (WHO, 1975, 1978, 1979a,b,c, 1980), national (Moore & Gerstein, 1981), and regional bodies (ARF, 1978) have advocated restrictions on availability as a preventive measure.

Interest in the regulation of availability is hardly a new or recent phenomenon. Many authors have documented historical and even ancient proscriptions regarding the distribution of alcohol (see, e.g., Robinson, 1977), dating as far back as the Code of Hammurabi around 2000 BC (Popham, 1978). The renewed attention placed on alcohol control measures since World War II is due to a number of factors. First, there has been a marked increase in alcohol consumption and in various indices of alcohol-related problems throughout the industrialized world (Bruun et al., 1975). At the same time, treatment systems appear to be limited in effectiveness (Edwards et al., 1977) despite their high cost. Thus further impetus is given to the search for preventive measures.

Another significant factor is the declining influence of the "disease concept" of alcoholism among public health professionals. In the postwar era, controls over the availability of alcohol were generally viewed as unpopular anachronisms dating from the earlier era, strongly associated with the failure of Prohibition (Davies & Walsh, 1983). Moralistic conceptions of drinking that were the basis of Prohibition were largely replaced by the "disease concept" of alcoholism. According to this viewpoint, normal social drinkers are fundamentally different from alcoholics, who are seen as suffering from a special disease. As noted in the report of the WHO-affiliated International Study of Alcohol Control Experiences (Mäkelä et al., 1981):

The locus of alcohol problems tended to be redefined from "the bottle" to "the man": if alcohol problems were a matter of specific defective individuals, then there was no need to control the drinking of the majority who were not defective. If, indeed, alcohol problems were specific to these individuals, an appropriate means of managing alcohol problems would be to provide treatment for them, and to try to persuade them — but not others — to abstain. In

this way, the expansion of the treatment system may be seen as a kind of cultural alibi for the normalization of drinking and relaxation of controls. (pp.64-65)

Because the unique predisposing factors that cause certain social drinkers to become alcoholics remained unknown, the conceptualization of alcoholism as a disease led to an emphasis on treatment rather than prevention.

More recently, the disease concept of alcoholism has been called to question for various reasons, including epidemiological research that shows that the risk of various adverse health effects increases significantly even for those who consume well below levels associated with alcoholism (Schmidt & Popham, 1975-76). For example, Péquignot and his colleagues (1974) found that even a relatively moderate intake of alcohol (between 60 and 80 g of ethanol per day) carries twice the risk of death from cirrhosis as the next lowest level of consumption (between 40 and 60 g). To put these numbers into perspective, 60 g of ethanol is equal to 4.4 "standard drinks," a standard drink being 12 ounces of 5% beer, or 5 ounces of 12% table wine, or a 1.5 ounce measure of 40% spirits. This level of consumption is far below that of clinical alcoholics (de Lint & Schmidt, 1968). Thus, even in "moderation," drinking is associated with the development of adverse consequences.

With the increases in alcohol-related problems in the 1960s and a declining enthusiasm for the disease concept of alcoholism among health professionals, interest began to focus on the potential of alcohol control measures. Room (1984) delineated three hypotheses concerning the general effects of controls over availability. The first is that controls over availability have no effects on alcohol problems, the second is that they have perverse effects, and the third is that they have positive effects.

The first argument, claiming no impact, was based mainly on *a priori* assumptions that alcohol-related problems stem primarily from alcoholics, who are unaffected by controls over access. Evidence severely challenges both of these assumptions. First, it cannot be assumed that all, or in some instances even most, alcohol problems can be attributed to alcoholics. Second, as we shall discuss shortly, alcoholics are, if anything, even more affected by controls over availability than social drinkers.

The second perspective claimed that controls over availability have negative effects and actually exacerbate alcohol problems (Chafetz, 1970; Wilkinson, 1970). It was argued that alcohol problems stem in large part from cultural ambivalence — a lack of adequate norms governing alcohol use, i.e., rules which would enable drinkers to distinguish properly between moderate and problematic drinking. It was therefore argued that preventive efforts should focus on providing normative rules regarding appropriate drinking behavior. According to this perspective, parents should introduce drinking to teenagers at home and restrictions on access to alcohol should be minimized to avoid the "forbidden fruit" attraction of alcohol.

This perspective has been discredited primarily on empirical grounds. Mäkelä (1975) critically evaluated the evidence for the perspective and found no support: variations in rates of alcoholism for cultural subgroups could largely be explained by variations in overall rates of drinking. Furthermore efforts to substitute moderate drinking practices for undesirable drinking

patterns have consistently failed in the absence of restrictions over access (Single, 1979).

The third line of argument is that controls over availability are negatively related to rates of alcohol problems and can therefore be used as a mechanism for the prevention of alcohol-related problems. The position is supported by considerable empirical evidence concerning the impact of availability on consumption levels and alcohol-related problems, to be summarized shortly. Additional support for this position derives from econometric studies on alcohol consumption. Contrary to what some persons might have expected given the addictive properties of alcohol and given the high proportion of consumption accounted for by heavy drinkers, alcoholic beverages have been found to be generally responsive to incremental changes in price (Österberg, 1975).

In 1975 an international group of alcohol researchers critically reviewed the evidence regarding the relationship between legal controls on alcohol availability, mean levels of consumption, and indices of alcohol-related problems (Bruun et al., 1975). To quote from their report:

Our main argument is well substantiated: *changes in the overall consumption of alcoholic beverages have a bearing on the health of the people in any society.* *Alcohol control measures can be used to limit consumption: thus, control of alcohol availability becomes a public health issue.* (pp.12-13)

In the decade that has passed since it first appeared, this influential report has been a central focus of a continuing policy debate. The controversial nature of the issue is reflected by the variety of terms used to describe the position that controls over alcohol availability can be employed to prevent alcohol-related problems. The 1975 international group referred to their position as "the public health approach" to alcoholism prevention. Unfortunately, this term has been used to describe somewhat different and more permissive approaches as well (see, e.g., Davies & Walsh, 1983). It also incorrectly implies a consensus among public health professionals regarding the desirability of alcohol controls.

At least one critic provocatively labelled the position as "neo-Prohibitionist" (Pittman, 1980). This is clearly inaccurate. The position does not advocate that alcohol be prohibited; nor is it based on the same reasoning as that of the Temperance interests that spawned Prohibition in many countries. Although Temperance groups and public health interests may on occasion both advocate greater restrictions on alcohol availability, the former base their proposals on moralistic conceptions regarding drinking, whereas the latter base theirs on epidemiological evidence regarding the consequences of alcohol use.

The position favoring the control of alcohol availability has also been termed the "single distribution" hypothesis or the "Ledermann hypothesis" (Schmidt & Popham, 1978), acknowledging the influence of the work of French scientist Sully Ledermann (1956) on the distribution of alcohol consumption. There is little dispute that the mean level of alcohol consumption in a society is closely related to numbers of persons consuming at levels deemed to be of high risk. There has never been documented a society with low

mean consumption that did not also have a relatively low number of "heavy" drinkers; and by the same token, societies with high levels of mean consumption tend to have high numbers of persons deemed to be "heavy" drinkers. Ledermann argued that a single function, the lognormal curve, provides an accurate estimate for the distribution of alcohol consumption in any society. Given the addictive properties of alcohol, some might have expected alcohol distribution to be bimodal, with one peak level among moderate or "social" drinkers and a second peak level among those who are physically dependent on alcohol. Contrary to this expectation, Ledermann found a unimodal distribution that is continuous and highly skewed.

Perhaps the most controversial aspect of Ledermann's work is the contention that the dispersion of this distribution is relatively invariate and can be estimated from the mean: that is, once the mean consumption among drinkers is known, one can predict with reasonable accuracy the number of persons consuming at any level, including the number who might be deemed to be "high risk" or "alcoholic." Bruun and his colleagues (1975) critically examined and extended Ledermann's analysis of the distribution of alcohol consumption, and their work largely supported this conclusion.

The contention that the Ledermann lognormal distribution represents an accurate description of alcohol consumption in any society has been challenged by many authors (Duffy, 1977; Duffy & Cohen, 1978; Parker & Harman, 1978; Pittman, 1980; Pittman and Strickland, 1981). Much of the criticism concerns assumptions underlying the development of the Ledermann distribution function, such as the assumption that the upper limit of alcohol consumption per person is 365 L of pure ethanol per year. More importantly, Skog (1982) has demonstrated several significant deviations from the Ledermann model.

One of the most perplexing issues with the Ledermann distribution is the assumption of homogeneity, whereby the model is only meant to apply to populations in which there are no major social groups that deviate substantially from the overall mean level of consumption. This assumption would seem to eliminate the model's applicability to any known society, for all societies consist of an approximately equal number of males and females, and male drinking levels are invariably a good deal higher than those of females. For example, survey data indicate that Canadian males drink at least 50% more per capita than females (Canada Health Survey, 1981). The Ledermann distribution would yield one estimate of the number of persons consuming 15 cL or more per day, given the total population of Canada, but a very different and considerably higher estimate if it were applied separately for males and females. In Ledermann's original formulation, a new parameter was added to the distribution to adjust for heterogeneous populations, but this very important aspect of the distribution function was ignored or omitted in Ledermann's subsequent work and in the work of others (an exception being Skog, 1982). Since no society meets the assumption of homogeneity, it would have been preferable if the single-parameter Ledermann function had been ignored instead.

The controversy surrounding the Ledermann hypothesis continues.

Skog, in particular, has carried on this line of work despite the fact that he views the Ledermann theory as "untenable" in its original form (1983, p.1). By modifying Ledermann's work to include a number of factors which combine in a multiplicative rather than an additive fashion, as well as considering the drinker's social network, Skog has transformed a theoretically crude empirical generality into a potentially viable theory concerning the etiology of alcoholism.

The purpose of this discussion is not to fully summarize the Ledermann controversy, as this has been done elsewhere (see, e.g., Davies & Walsh, 1983; Skog, 1982). Rather, the intent is to point out that the policy debate surrounding the feasibility of controlling alcohol availability as a means of preventing alcohol-related problems has focused on a relatively narrow aspect of the theoretical connection between availability and problems *that is not at all a necessary, or integral, part of the argument favoring controls*. As we shall see, the Ledermann hypothesis is only one of several possible explanations for the observation that the mean level of consumption in a society is related to the number of heavy drinkers, and this relationship is by no means the only connection between alcohol and availability and alcohol-related problems.

Thus, the Ledermann hypothesis has received undue attention. It is not the cornerstone for the position that control over alcohol availability can be used as a preventive measure. For this reason it is preferable to use the less controversial and more descriptive term "availability theory" to refer to the set of hypotheses that postulate that controls over the availability of alcoholic beverages influence the incidence of alcohol-related problems.

4. CHARACTERISTIC RESEARCH METHODS

As discussed earlier, the dependent variable in the availability theory is not "alcoholism" but "alcohol-related problems." This focus stems from difficulties in the conceptualization and operationalization of the concept of "alcoholism." A brief discussion of these definitional concerns is illustrative of how changes in the conceptualization of alcohol problems have a marked impact on the debate surrounding the availability theory.

In the first place, there is very little consensus as to what exactly is meant by the term "alcoholism." Jellinek's classical description of the alcoholic involved a progressive development of well-defined phases and symptoms (Jellinek, 1947). This conceptualization was based mainly on populations of clinical alcoholics in the United States. As several authors have pointed out (see, e.g., Room, 1977), since the time of Jellinek's formulation evidence has come to light indicating that the extent and nature of alcohol-related problems are quite different in general populations as compared with clinical alcoholics. For example, most clinical alcoholics are well past 30 years old, whereas in general population surveys it is men under the age of 30 who consistently score the highest on various indices of problem drinking (Room, 1977).

There is also a major discrepancy with respect to the number of problem drinkers identified in surveys and the number of clinical alcoholics. Edwards

(1973) found that the number of persons defined as "problem drinkers" in a general population survey was nine times the number of persons identified as alcoholics by clinical or agency data. Room has interpreted these findings as indicating that the problem of the "alcoholic" is in part due to a labelling process by which heavy drinking patterns that are tolerated among young men are deemed inappropriate among the relatively few middle-aged drinkers "who retain their youthful heedlessness and drinking style" (Room, 1977, p.83).

Given the existence of at least two distinct sets of problem drinkers — a young, untreated group in the general population and a special group of older, chronic drinkers typical of those found in treatment settings — it is not surprising that there are a wide variety of methods used to estimate numbers of "alcoholics." The prevalence of alcoholism has been measured by treatment data (Lau, 1978; Walsh & Walsh, 1973), survey techniques (Bailey et al., 1965; Cahalan et al., 1969; Cahalan & Room, 1974; Manis & Lunt, 1957; Moskalewicz & Wald, 1979; Mulford & Miller, 1959-60; Müller, 1979; Simpura, 1979), consumption data derived from aggregate sales or tax records (de Lint & Schmidt, 1968; Ledermann, 1956; Schmidt & de Lint, 1970; Single & Giesbrecht, 1978), and from data on alcohol-related mortality (Cameron, 1979; Celentano, 1976; Jellinek, 1947; Jellinek & Keller, 1952; Keller & Efron, 1955; Schmidt & de Lint, 1970; Single, 1979).

Each of these methods has its advantages and disadvantages. Treatment data are frequently more an indicator of availability of facilities than of alcoholism prevalence. The indirect measure of alcoholism prevalence based on consumption data (Ledermann, 1956) attempts to estimate the number of persons with rates of consumption similar to those of clinical alcoholics, in accordance with the theory that aggregate consumption data enables one to predict the distribution of consumption in a society. The indirect measures based on mortality data, such as the Jellinek estimation formula (1947), similarly use data concerning the incidence of alcohol-related mortality among clinical alcoholics to generate an estimate of the total number of alcoholics in a society.

The raw data in indirect estimation procedures are typically statistics that are routinely collected. Thus, the use of these methods is relatively inexpensive. Further, the fact that the results of various indirect estimation procedures tend to be roughly consistent with one another thereby enhances confidence in the results. Schmidt and de Lint (1970) examined four alternative indirect estimates of the number of alcoholics in Ontario in 1966 and found a close correspondence in the estimates. Single (1979) replicated the study using 1973 data and obtained the same results.

Despite these advantages, there are a number of drawbacks to the use of indirect estimation procedures. First, these methods can only be used to estimate the number of "alcoholics" in a given society at a particular time. They do not identify specific individuals and therefore they do not provide data on the correlates of alcoholism. Secondly, the alcoholic population that is being estimated may be too narrowly defined. The Ledermann procedure (1956) attempts to estimate the number of persons consuming alcohol at levels

equal to the minimal levels among clinical alcoholics. Many such persons may not have any social or behavioral problems associated with alcohol use; and by the same token, there are undoubtedly many persons who consume lower amounts of alcohol who experience alcohol-related problems. The well-known Jellinek estimation procedure (Jellinek, 1947; Jellinek & Keller, 1952) similarly assumes the clinical alcoholic to be typical of all alcoholics, in that certain key elements in the estimation formula are derived from studies on clinical populations.

Further, both the Ledermann and Jellinek formulae have been criticized on technical grounds. As noted earlier, the Ledermann equation has been criticized frequently for not providing an adequately close fit to the true distribution of alcohol consumption (see, e.g., Parker & Harman, 1978; Skog, 1982). In the original formulation of the Jellinek procedure, the rate of death due to cirrhosis among alcoholics and the proportion of all cirrhosis deaths attributable to alcoholism were treated as constants (Jellinek, 1947). Popham (1970) and others have noted that these rates are in fact variable. This criticism does not, however, necessarily invalidate the Jellinek formula. Rather, it indicates the need to collect independent data on the requisite rates so that they are reasonably current and appropriate to the particular setting in which the formula is applied.

There is an extensive literature on the use of surveys to measure individual levels of alcohol consumption and drinking problems (see Room, 1977). In surveys, problem consumption is generally operationalized in terms of a high self-reported intake, via a quantity-frequency scale (e.g., Straus & Bacon, 1953). Alternatively, the researcher may use a set of questions intended to tap the basic behavioral and social problems associated with "alcoholism." Such problems vary according to the setting and the characteristics of the drinker, and thus it is virtually impossible to develop a measure that would be reliable in every setting at any time. The use of a quantity-frequency scale suffers from an even more serious problem: namely, that typically alcohol consumption is highly underestimated in surveys (Mäkelä, 1969; Pernanen, 1974; Room, 1971). Further, the extent of underreporting is not random but strongly related to volume of intake (Schmidt, 1973).

Given these problems, it is perhaps not surprising that various survey methods have provided somewhat discrepant results. Celentano (1976) examined five different indicators of alcoholism from survey data in Washington County, Maryland, and found very divergent results, ranging from a rate of 3.1% on the Mulford and Miller Social Problems Scales to 15.2% on a revised version of the Strauss and Bacon quantity-frequency scale. Despite their limitations, survey techniques remain a common method for ascertaining the nature and extent of alcohol-related problems. A major advantage of survey methods relative to indirect estimates of alcoholism prevalence is that surveys provide data on individuals, thus enabling the researcher to correlate individual characteristics with propensity toward alcohol-related problems or heavy alcohol consumption.

In short, no single estimate of the prevalence of alcoholism is entirely satisfactory. The preferred approach is to examine a variety of indicators; if

different indicators provide reasonably consistent estimates, confidence in the results is enhanced. The use of multiple estimates may also provide information on the differential manifestation of alcohol-related problems (Single, 1979).

In summary, the availability theory is not limited to alcoholism. Rather, it is concerned with a wide variety of chronic *and* acute problems as well as health *and* social problems associated with alcohol use. On the other hand, the theory is very much a limited one in that, unlike a grand theory, it does not attempt to explain all or even a major portion of variation in individual or societal rates of alcohol-related problems. It merely holds that availability is significantly related to alcohol problems. Access to alcohol is not necessarily the sole or even the primary determinant of problems.

5. RESEARCH RESULTS

To this point, it has been argued that contrary to what some authors have asserted (see, e.g., Davies & Walsh, 1983; pp.2-3), the availability theory does not derive from nor depend on any particular hypothesis concerning the distribution of alcohol consumption. The cornerstone of the theory is not the Ledermann lognormal distribution function, but rather the empirical relationship of control measures to alcohol consumption and to alcohol-related problems. A brief summary of research on these issues is presented below; for more thorough reviews, see Farrell (1985), Moskowitz (1987), Popham et al. (1976), and Smith (1983).

5.1 TYPE OF CONTROL SYSTEMS

There is enormous variability in the general approach to alcohol control in different societies. Prohibition is not a historical anachronism; it is common among Moslem countries today and there are even regions within Western countries such as Canada where the sale of alcohol is proscribed by law. On the other hand, many countries have an essentially laissez-faire approach typical of the wine-producing countries of the Mediterranean. In Nordic countries such as Finland, both production and distribution are in the hands of a state monopoly. In Canada and several states in the U.S. the distribution of alcohol is regulated by a state monopoly but production is privately owned. In other states in the U.S. and most Western European countries, both production and distribution are in the hands of privately owned business under licence by the government.

There is little doubt that regardless of other consequences, total prohibition does succeed in reducing alcohol consumption and alcohol-related problems. Those countries that enacted prohibition laws in the early part of this century, and areas subjected to de facto prohibition due to war, experienced substantial reductions in health and social problems associated with alcohol use (Bruun et al., 1975).

The evidence regarding the impact of less dramatic differences in alcohol control systems is not conclusive. Monopoly systems do not necessarily have

lower rates of alcohol consumption than private systems. By the same token there are laissez faire systems with relatively low consumption, the Netherlands being an example (de Lint, 1981). In the U.S., there is little difference between those states that have a monopoly system and those with a private licence system in terms of alcohol sales or rates of cirrhosis (Popham et al., 1976).

Comparisons between societies with different types of control regimes are necessarily inconclusive, regardless of the findings, because of confounding influence of alcohol culture. Changes in the nature and extent of controls over time within particular societies indicate that the liberalization of controls that has occurred since World War II throughout the industrialized world has been accompanied by an increase in alcohol consumption and increases in indicators of alcohol-related problems (Bruun et al., 1975). As noted earlier, it is not always clear whether increased consumption is an effect, a cause, or a concomitant of liberalization of the alcohol laws. It is also not clear whether the apparent negative relationship between consumption and controls would hold true if greater restrictions were put in place.

5.2 RESTRICTIONS ON DISTRIBUTION: HOURS OF OPERATION, DAYS OF SALE, AGE LIMITATIONS, AND ADVERTISING

Increased access to alcohol has generally been found to be related to increased consumption. When local prohibition was ended in selected towns in rural Finland, there was a dramatic increase in alcohol consumption and alcohol-related problems (Kuusi, 1957). Controls on hours of operation of alcohol outlets have been found in a number of settings to be related to consumption patterns and alcohol problems. Popham (1982) found arrests for public drunkenness to be correlated with hours of tavern operation in Toronto. In Australia, tavern hours were changed in an attempt to reduce the problems associated with a 6 p.m. closing time. Workers had been drinking large quantities so rapidly during the brief time after work when drink was available that the time immediately prior to closing was known as the "six o'clock swill." When the closing time was extended to 10 p.m. there was a corresponding change in the temporal pattern of traffic accidents, but overall consumption was not clearly affected (Raymond, 1969).

The findings regarding the impact of age restrictions are unequivocal. The lower the drinking age, the lower the age of first use of alcohol, the higher the consumption of alcohol and the higher the indices of alcohol-related problems in the population at large, particularly teenagers (Douglass & Filkins, 1974). A case in point was the situation in Ontario, where the drinking age was lowered to 18 from 21 in 1971, but subsequently raised to 19 in 1978 as a result of increased drinking problems among teenagers, particularly a sharp rise in teenage drinking/driving accidents (Single et al., 1981, p.152).

One further aspect of distribution is more concerned with demand than supply, but nonetheless bears mentioning. A recurrent issue in the alcohol policy debate is the impact of advertising alcoholic beverages. The industry has frequently claimed that advertising only influences brand preferences and that there is no demonstrable impact on overall consumption. On the other hand,

public health professionals have taken it as almost self-evident that the enormous amount of money spent on advertising affects overall consumption, particularly when the advertising is aimed at new markets such as youth or women. Until recently, there was a dearth of comprehensive studies on the impact of advertising. Some research supports the industry claim that advertising only affects brand preferences (see, e.g., Bourgeois & Barnes, 1979), but these studies were generally based on ecological analyses involving correlation between alcohol sales and advertising expenditures in geographic areas. More recent work, based on a variety of research designs including data on individual consumption, has concluded that advertising may have an effect on consumption (Atkin & Block, 1980).

5.3 DENSITY OF OUTLETS

There have been a number of recent reviews of the research literature concerning the relationship of the number of outlets to alcohol consumption and to alcohol problem indicators (Farrell, 1985; Macdonald & Whitehead, 1983; Smith, 1983), all of which arrive at the same basic conclusion. As stated by Macdonald and Whitehead (1983):

The weight of evidence, especially when one takes into account the quality of the studies, is on the side of the availability of outlets accounting for some of the variance in the extent of alcohol consumption. The availability of outlets for off-premise consumption appears to be more sensitive than the availability of on-premise consumption, but the impact of the latter cannot be treated as trivial without further study. (p.482)

Smith (1983) summarized the research with the following points:

- While giving little or no attention to possible confounding variables a number of "early" studies gave insignificant results.
- The introduction of on-premise drinking, when off-premise drinking was already permitted, did not result in any significant effects. A similar comment is also applied to the liberalization of on-premise drinking.
- The introduction of extension of liquor licences for off-premise sales by grocery stores or supermarkets possibly results in increased consumption of wine and more convictions for drunkenness.
- A number of North American studies have provided evidence of a positive relationship between the number of liquor stores and consumption of spirits, wine, and alcohol-related problems. However, the above findings cannot be generalized to packaged beer as liquor stores in North America sell primarily wine and spirits.
- The number of licences for on-premise drinking appear to be positively related to a variety of indices for the use and abuse of alcohol. However, on the basis of only two studies the relationship should be regarded as tentative.
- Factor analytic studies suggest that on-premise availability and off-premise availability bear little relationship to each other. However, as can be seen above, each appears to be positively related to various indices for the use and abuse of alcohol. This may explain why the results of studies in which variables for the total number of outlets have been used have also been positive.
- Virtually all the studies were conducted in North America
- The problem of determining the direction of the apparent causation was commented on by a number of researchers. Does increased availability cause

the increased use and abuse of alcohol, or does the demand for more alcohol lead to greater availability? (p.88)

The available evidence from Ontario is limited to correlational analyses, based on county data, but the results serve to illustrate these findings. Rush and his colleagues (1986) found both on-premise and off-premise density of outlets to be related to consumption and problem indicators. Unfortunately, correlations convey relatively little intuitive understanding. Such statistics are useful for determining *whether* an independent variable makes a difference, but they convey little sense of *how much* of a difference it makes. Therefore, the raw data presented in Rush et al. (1986) have been reanalysed in simple tabular form and are presented in Table 1.

It should be noted that Table 1 does not simply present the selected consumption and problem statistics by outlet density because the term "outlet density" can be misleading. Many persons would think that urban areas such as Toronto have a high density of outlets, and that rural areas, such as in Northern Ontario, have a low density. If outlet density is operationalized as the number of outlets per unit of population, however, one finds just the opposite. Metropolitan Toronto in fact had the *lowest* density of outlets per unit of population (.52 per 1000 adults), whereas remote sections of the province tended to have the *highest* density of outlets. One could instead attempt to compute a measure of outlet density based on the number of outlets per unit of territory in a county, but this figure would also be less than adequate because many counties contain enormous tracts of uninhabited land and would be artificially underscored on outlet density. Ideally, one would prefer a measure that considers the mean distance between residences and the nearest outlets. Unfortunately, this calculation is impractical.

In view of these difficulties, Table 1 presents a three-variable table, showing differences in alcohol consumption and alcohol problems between counties with above-average number of outlets per unit of population and below-average number of outlets per unit of population, with the degree of urbanization controlled. Thus, from the first two columns of the table, it can be seen that among rural counties (those with less than half of the population living in communities of 2,000 or more), consumption rates are much higher and problem rates are somewhat higher in those counties that have a greater than average density of outlets. The same findings hold true for mixed and urban counties. These Ontario data corroborate the general pattern found in other jurisdictions — the number of outlets is positively related to rates of alcohol consumption and alcohol-related problems.

5.4 PRICING AND TAXATION

One of the most important aspects of availability is the ability of the consumer to afford alcoholic beverages. As noted earlier, alcohol consumption is "price elastic": that is, responsive to incremental changes in price. The econometric studies of price elasticities have been corroborated by experimental research that has found that reducing the prices of alcoholic beverages during "happy hours" significantly increases consumption among both light and heavy drinkers (Babor et al., 1978). These studies are also consonant with

TABLE 1

Alcohol consumption and selected problem statistics by urbanization and outlet density per unit of population

	URBANIZATION: % OF POPULATION LIVING IN COMMUNITIES OF 2000 OR MORE			
	Rural: 50% or less	Mixed: 50%-75%	Urban: 75% or more	
# outlets above average	# outlets below average	# outlets above average	# outlets below average	# outlets above average
Consumption, per capita, litres of absolute alcohol	15.15	10.26	13.31	10.70
Cirrhosis mortality, rate per 1000 adult deaths	.22	.15	.22	.15
Traffic mortality, rate per 1000 adult deaths	2.04	2.01	1.94	1.92
Cirrhosis morbidity, rate per 1000 hospital separations (Number of counties*)	.79	.73	1.07	.75
	(9)	(7)	(4)	(15)
				(2)
				(12)

* The consumption, morbidity, and mortality rates are county averages, not weighted by population size. They do not represent individually based data. Thus, for example, the first row presents the mean county rates of alcohol consumption for each type of county, not the average individual consumption in each type of county.

Source: Reconstructed from data presented in Rush et al. (1988).

previous findings concerning the impact of differential price increases on patterns of consumption in a number of situations. In both Belgium (Reuss, 1959) and Denmark (Neilson & Stromgren, 1969) exceptionally high taxes on spirits led to marked reductions in consumption of spirits. More recently, the consumption of fortified wine was successfully reduced in Finland and in Ontario (Single et al., 1981) by the introduction of substantial price increases.

The use of alcohol taxation is a very promising mechanism for the prevention of alcohol-related problems. Indeed, a study of the impact of liquor taxes on alcoholism commissioned by a special alcohol policy panel under the auspices of the U.S. National Academy of Sciences estimated that "an increase in the liquor excise tax of one dollar per proof gallon reduces the liver cirrhosis mortality rate by 5.4% in the short run and by perhaps twice that amount in the long run" (Cook & Tauchen, 1981, p.2). Although these findings do not necessarily apply elsewhere, they are indicative of the potential that alcohol taxation policy has as a tool for the prevention of alcohol problems.

5.5 DIFFERENTIAL IMPACT ON "SOCIAL" VERSUS "HEAVY" DRINKERS

All of the aforementioned research on the impact of controls over availability would provide only weak support for their use as a preventive measure if it were found that these controls only affected moderate or infrequent drinkers and had relatively little effect on high volume or "heavy" drinkers. However, evidence had gradually accumulated that controls over alcohol availability have an even greater impact on high risk or "heavy" drinkers than on moderate drinkers.

Room (1984) has summarized this evidence, which stems mainly from econometric studies on the impact of price changes and from a rich literature on the impact of strikes by alcohol workers. As Room comments: "Though the strikes often attracted extraordinary popular attention, and the population as a whole often substantially shifted its choices of beverage and drinking locale, the amount of consumption of moderate drinkers, particularly those of middle class, was often little affected. On the other hand, the strikes often had dramatic effects in reducing alcohol-related problem rates associated with poor habitual heavy drinkers" (Room, 1984: 309). Room concludes that it is no longer tenable to claim that we lack studies linking decreased availability with decreased consumption by heavy consumers of alcohol.

6. PRACTICAL IMPLICATIONS AND RELATIONSHIP TO OTHER THEORIES

In sum, the availability theory holds that access to alcoholic beverages will be positively related to levels of consumption by both moderate and heavy drinkers, and, consequently, availability will influence indices of alcohol-related problems. The theory focuses on a wide array of chronic and acute health and social problems associated with both moderate and heavy alcohol use, rather than on alcoholism *per se*. A number of important caveats have been noted, including the warning that the theory makes no claim to causal priority.

The availability of alcohol is not viewed as the sole or even the most important factor determining an individual's risk of suffering adverse consequences from drinking. However, unlike most of the other determinants of alcohol-related problems, which are beyond the reach of public policy, availability can be manipulated as a mechanism for problem prevention.

The basic way in which availability has been linked to alcohol problems is via a set of three interrelated hypotheses: availability influences mean consumption levels, which influence heavy drinking, which in turn is related to adverse health and social consequences. However, there are a number of other linkages between availability and alcohol problems. Indeed, undue attention has been placed on the Ledermann or "single distribution" hypothesis concerning the distribution of alcohol consumption. The Ledermann hypothesis is not necessary to the availability theory.

The practical implications of the availability theory are almost self-evident. If access to alcohol can influence rates of consumption and alcohol problems, restrictions on access can be used as a preventive mechanism. Indeed, as noted earlier, the theory developed in large measure as a response to demands for policy recommendations, and it has been used as the cornerstone for public health representations regarding alcohol policy to governments at the provincial, national, and international levels.

Whereas the availability theory is of enormous and obvious practical policy relevance, it has tenuous connections at best to other theories. It does not derive from an established body of theory in criminology, social psychology, sociology, or other social science. Its genesis is more empirical than theoretical: it developed from the recurring need to find mechanisms to prevent alcohol problems rather than from the application of a larger theory to alcohol policy issues.

Perhaps the one set of larger theories with which availability theory is most closely linked is the economic theory of consumer behavior (see Godfrey & Maynard's chapter in this volume). As mentioned earlier, alcoholic beverages, like other commodities, tend to be responsive to changes in price. In a comprehensive review of studies of the price elasticities of alcoholic beverages, Finnish economist Esa Österberg (1975) stated:

Econometric analyses based on the classical theory of consumer demand and relating to different countries and different periods have shown that fluctuations in the demand for alcoholic beverages can by and large be explained statistically in terms of prices, incomes and consumer preferences — that is, in terms of the same variables as are involved in the fluctuations of demand for other commodities. Alcoholic drinks appear therefore to behave on the market like other commodities and in the way presupposed by the theory of consumer demand. (p.2)

Thus, although the availability theory did not derive from economic theory of consumer behavior, it is certainly consistent with it. There are other well-grounded bodies of theory that may prove to be relevant to the availability theory. Future work in this area should focus on the less well studied connections between availability and alcohol problems, and on the influence of the individual's social network and situational determinants of drinking.

behavior. In this manner, the connection between moderate and heavy drinking would no longer be viewed as a matter of statistical distribution, but rather as the complex, dynamic social process it surely must be. Skog's works on risk functions (Skog, 1984) and social networks (Skog, 1986) are important first steps in this direction.

7. FUTURE DIRECTIONS FOR RESEARCH ON AVAILABILITY

Thus far research on alcohol availability has concentrated on the relationship between controls and consumption or problem rates. As the weight of evidence grows that controls do impact on alcohol consumption, it should be expected that future research will focus on other aspects of availability as they affect alcohol consumption and problems. I expect that greater attention will be paid to the following aspects of availability: (1) the impact of serving staff and training programs aimed at promoting responsible beverage service; and (2) the structure of the alcohol industry as it affects the availability and consumption of alcohol.

7.1 CIVIL LIABILITY AND SERVER TRAINING PROGRAMS

Less than fifteen minutes after finishing his last beer and leaving the Arlington House Hotel, Clayton Sharpe failed to negotiate a curve in the road. His car plunged over a steep embankment, rolled over repeatedly, and came to rest 124 meters away. Sharpe suffered only minor injuries, but his passenger, 16-year-old Andreas Schmidt, was critically injured. Although he survived, Schmidt was rendered a quadriplegic.

Schmidt and his family sued not only Sharpe, but also the Arlington House Hotel for \$13,000,000. After a lengthy trial, judgment was awarded against both defendants for over \$1,390,000...

As discouraging as it may be to Canadian tavern owners and their insurers, the Schmidt case cannot be written off as a legal aberration. The clear trend in the law is toward the expansion of liability for all those who sell or supply alcohol to others. (Solomon et al., 1985, p.257)

The Arlington House Hotel was sued in the Schmidt case as a provider of alcohol for breaching an obligation to control the conduct of its intoxicated patrons. There is a distinct trend in the U.S. and British Commonwealth countries, toward the expansion of civil liability of providers of alcohol. This liability may be based on statute, as is the case in three Canadian provinces and those U.S. states with "dram shop" laws, it can be based on common law, or it may be based on both. In addition to their liability as providers of alcohol, tavern owners and social hosts are further liable for the conduct of intoxicated guests as occupiers under statutory occupier liability in virtually all jurisdictions.

The factors responsible for the recent emergence and expansion of civil liability of the intoxicated are: (1) an expansion of the duties of affirmative action to control the conduct of others in a wide variety of circumstances; (2) the development of mandatory breath and blood testing, making it easier to

establish intoxication as a cause of injury in court cases; (3) the narrowing of traditional defenses such as the voluntary assumption of risk, such that now courts will not accept a claim that a drunk driver or his or her passengers voluntarily accepted liability for their actions unless there was an explicit agreement to do so; (4) increases in the size of damage awards, due to changes in methods of determining awards and advances in medical technology which enable many seriously injured accident victims to survive; and (5) changing public and judicial attitudes which are much less accepting of drunk driving (Solomon & Single, 1986).

The hospitality industry has responded to the increased threat of civil liability in a generally positive and constructive manner, developing programs aimed at preventing problems which may give rise to liability suits. Practices and policies which reduce liability risks include insuring the safety of premises, controlling access, promoting the consumption of food with alcohol, offering nonalcoholic or low-alcohol alternatives, setting drinking limits for patrons, and training staff in responsible serving practices.

The available evidence from tavern studies indicates that serving practices have a marked impact on drinking behavior. "Waiters and bartenders grant regular status to patrons via friendship behaviour, protective functions, special privileges and other preferential treatment. Their influence is so strong that it may be claimed that they select regular patrons and determine drinking rates" (Single, 1985, p. 17). Therefore, the widespread training of servers has considerable potential for the prevention of problems arising from acute intoxication, particularly in licensed establishments.

At least one U.S. state, Oregon, and two Canadian provinces, Ontario and British Columbia, have developed a policy of mandatory server training for all persons employed in the hospitality industry. The development and evaluation of both mandatory and voluntary server training programs represents a promising area for future research on alcohol availability.

7.2 THE STRUCTURE OF THE ALCOHOL INDUSTRY

Finally, there is a further aspect of availability which has been neglected in the past and which may be given greater attention in the future: studies on the structure of the alcohol production and distribution industries. Two recent World Health Organization projects (WHO, 1982; WHO, 1987) have addressed this topic, and the WHO has repeatedly expressed concern over the excessive marketing and promotion of alcohol beverages in the Third World on the part of private producers and monopolies from developed countries. These projects spawned a number of studies on particular aspects of alcohol distribution; thus, for example, Casswell and Smythe (1982) documented the excessive promotion of alcohol by multinational corporations in five South Pacific societies.

An analysis of the intercorporate ownership and interlocking directorates of the alcohol industry in Canada (Single, 1987) yielded several findings which bear upon alcohol availability. It was found that ownership is highly complex: the alcohol industry has been generally transformed from a large set of small firms owned by entrepreneurs to a small set of large international

corporations with extensive and complex intercorporate connections. The distilling and brewing industries in particular have become highly oligopolistic.

From a public health perspective, this has had both positive and negative effects on alcohol availability. On one hand, the industry structure facilitates large advertising expenditures, the development of new products, and the promotion of consumption in other ways as well. On the other hand, oligopolistic pricing and high barriers of entry in the market have restricted competition and thus avoided certain excesses associated with fierce competition.

8. CONCLUSION: FUTURE DIRECTIONS REGARDING, POLICY ISSUES

The future regarding the policy issues addressed by the availability theory is not clear. The general trend throughout the industrialized world since World War II has been marked by the liberalization of alcohol controls, increased alcohol consumption, and increases in indices of alcohol-related health and social problems (Bruun et al., 1975; Mäkelä et al., 1981). As stated earlier, the predominance of the disease concept of alcoholism and the expansion of the treatment system have served to facilitate the trend toward liberalized controls.

In the report of the International Study of Alcohol Control Experiences, it was noted that there are some indications of change in this response to drinking problems (Mäkelä et al., 1981). Not only is there increasing skepticism about the disease concept of alcoholism, but many societies have been faced with escalating public welfare expenditures in the context of economic contraction. The need to reduce expenditures in the health and welfare sector has forced policy-makers to consider preventive measures as alternatives to relatively expensive treatment modalities. Thus, the use of controls over alcohol availability as a preventive mechanism has been receiving greater attention than it has in the past.

Regardless of its motivation, serious consideration of controls is a welcome development. In the past policies concerning the availability of alcohol were generally viewed in strictly economic terms. There is clearly a need to infuse consideration of the consequences to the public health of decisions concerning the availability of alcoholic beverages. If the availability theory is correct, then availability of alcohol is more than an economic issue—it is also a public health issue.

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10. EMERGING ANTHROPOLOGICAL THEORY AND MODELS OF ALCOHOL USE AND ALCOHOLISM

Dwight B. Heath

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INTRODUCTION

The cornerstone of all anthropological contributions to the understanding of alcohol use and its outcomes is an emphasis on social and cultural factors. However fundamental the chemistry of a beverage may be, and however variable the biology and psychology of an individual drinker, detailed familiarity with those factors — and with their metabolic, pharmacological, physiological, and other ramifications — will still leave out crucial factors that must be considered if we are to understand how the beverage and the organism interact. It has become customary in recent years to give at least lip service to recognizing the multifactorial nature of alcohol-related problems; acknowledgment is epitomized in the characterization of alcoholism as “a biopsychosocial disease,” of which the “social” component is dealt with primarily by anthropologists and sociologists.

There is a strict sense in which it could be said that there is no anthropological theory of alcoholism. One reason is that few anthropologists focus their attention on alcoholism, although many have a broader interest in

the wide range of beliefs and behavior that are associated with alcohol, including problem-free drinking as well as various alcohol-related problems. Another reason is that the several propositions and concepts on which anthropological studies of alcohol are grounded have rarely been articulated as an explicit theory, if one uses that term in the narrow and precise sense in which some philosophers of science do. However, if one follows normal usage of the term "theory," as most people do, it is evident that there are several propositions and models grounded in anthropology and sociology that have immediate relevance to alcoholism and that can be said to constitute significant parts of an emerging sociocultural theory of alcohol use and its outcomes. It is especially noteworthy that such social and cultural models are some of the most popular, influential, and comprehensive among the many types reviewed in this volume. The reason for this interplay of diverse parts, many of which are well known and respected, interacting with a core concept that has rarely been explicitly considered, is a fascinating paradox that comprises a complex but revealing study in both the philosophy of science and our understanding of alcohol. In this paper, I will address that paradox, and discuss what appears to be the emerging anthropological theory about alcohol use.

Anthropology, the science of humankind, is generally conceived as an immensely broad discipline. At many universities throughout North America — and in a few other places — it embraces a number of distinctive subdisciplines. These include cultural or social anthropology (often linked, in recent years, as sociocultural anthropology), which deals with patterns of belief and behavior among various populations; archeology focuses on populations in the past, whether prehistoric or historic; and physical anthropology is the study of human biology, past and present.

Even within the ample realm of sociocultural anthropology, linguistics is often differentiated as a separate discipline; so are folklore, social psychology, and various other fields. During the past couple of decades, there has been a further proliferation of topical subdisciplines emphasizing particular aspects of culture, such as medical anthropology, economic anthropology, political anthropology, and so forth. A few specific methodological approaches have earned recognition as fields of study, such as symbolism, ethnosemantics, holocultural studies, and others.

Although the use of alcoholic beverages can have interesting and important implications in any or all of the several branches of anthropology, as described elsewhere by Heath (1975, 1986a,b, 1987a), the present chapter will focus on theories and models that derive from the data and approaches of sociocultural anthropology and sociology. Within sociocultural anthropology, ethnography can be characterized roughly as the detailed study of ways of life of particular populations; ethnology, at a similar level of generality, is the study of similarities and differences that are found in various ethnographic studies. Sociology often involves the use of very different methods of investigation and analysis, but its subject-matter often coincides, at least in part, with that of sociocultural anthropology. Prior to World War II, it was commonplace that sociologists studied their own societies, usually those in the mainstream of Western cultural history, whereas anthropologists generally studied other

societies, usually non-Western in tradition. This division of labor has been eroded as members of both disciplines have shifted their foci of study. Many sociologists now deal with alien cultures, while some anthropologists study modern systems in complex urban settings. For purposes of this review of theories about alcohol use, however, it seems appropriate to discuss the contributions of scholars from various disciplines; the common thread is their focus on socially shared beliefs and behaviors with respect to alcoholic beverages.

It is remarkable that anthropology and sociology, fields in which the study of alcohol has not played a major role, have nonetheless produced such a plethora of models of alcohol use. Similarly, the fact that so many of those models are in the intermediate range, useful in both practical and conceptual terms, is unusual in these two fields in which theorizing has tended either to be ignored or to be on such a macroscopic scale as to have little immediate applicability. Another remarkable feature is the popularity and widespread acceptance that many of the models have been afforded, even by people whose focus of interest does not normally encompass social and cultural factors (e.g., DeLuca, 1981; Royce, 1981). Yet another extraordinary feature of these models is their predominantly inductive quality: in only a very few instances have social scientists set out to collect data specifically in order to test a particular theory; much more often, the importance of data on alcohol-related beliefs and behavior became evident only after the data had been collected. Furthermore, many of these models have the advantage that they can be applied, with slight modification, to the understanding of risk factors in individuals and also to gross differences among populations, measured in epidemiological terms and reported as differential rates of prevalence. All in all, the principal contributions of anthropological and sociological models are impressive, especially in view of the fact that no social scientist has ever appeared to be concerned, in a consistent and systematic way, with delineating such models as the major focus of his or her scholarly efforts.

✓ 2. PRINCIPAL ELEMENTS OF AN EMERGING ANTHROPOLOGICAL THEORY

Within sociocultural anthropology and sociology, there are several kinds of dialectic, or contrasting oppositions, that influence methods, data, and theories. One of these has to do with the dual concern for understanding both the minutiae of specific actions and events and also the principles that underlie general themes, institutions, and processes. On the one hand, social scientists often pride themselves on the special strength that comes from "a holistic approach" — that is, attempting to understand any specific sociocultural system in its own terms, as an integrated whole that is a unique agglomeration of attitudes, values, matériel, social relationships, and other traits that can only be understood as they relate to each other, and that have special levels of meaning if one can ascertain the views held by "insiders." On the other hand, students of social systems often pride themselves on the special strength that

comes from "a comparative approach"—that is, attempting to understand social and cultural phenomena and dynamics in a broad sense, trying to identify and analyse the features of human thought and action that distinguish it from other orders of life, looking for laws—or at least regularities—in the human experience that cut across time, space, and the boundaries of different kinds of groups that are important to their members.

Another of the dialectics that pervades the social and behavioral sciences is the concern with relating how and why any given person behaves as he or she does, to details about how and why classes, ethnic or religious groups, institutions, and other collective, corporate, or social phenomena behave as they do.

Although it may appear to reveal a weakness of the discipline, the fact that anthropologists have not often invested much time, thought, and effort in refining a theory of alcoholism may, on the contrary, reflect one of its special strengths, inasmuch as data are often reported in sufficient detail that they can be used for alternative interpretations and even for transdisciplinary theorizing in the future. One crucial reason for this apparent anomaly is the fact that anthropologists have, with only a few exceptions, paid very little attention to alcoholism. Although alcohol is one of the most widely used psychoactive drugs in the world, and also the one that has been used longest by humankind, the idea that it is associated with problems is absent in many cultures, and the idea that it might be a major factor in the etiology of a debilitating disease is highly unusual. There is a large and diverse literature on drinking and its sequelae, but the emphasis is more often on patterns that are normal in the given sociocultural context, on evaluative and emotional concomitants, and on functions that drinking and/or drunkenness seem to serve either from the point of view of the actors themselves or in terms of analysis of social systems.

Another reason that could be cited to explain the paucity of anthropological theories on alcoholism is that to date most of the sociocultural work on alcohol has been empirically grounded, with interpretations emerging inductively from descriptions that were made by naive observers (not gullible or ingenuous, but free of preconceptions).*

With those parameters in mind, it is all the more gratifying that so many of the findings of anthropology have come to enjoy widespread acceptance, and are afforded major importance, both by a broad cross-section of the lay public and also among many colleagues in that peculiar multidisciplinary field of theoretic and practical concerns that has come to be called "alcohol studies."

Public health models of various disorders are often phrased in terms of agent, host, and environment (or, with respect specifically to drugs: drug, set, and setting). In the case of alcoholism, the agent (or drug) is alcohol (normally ethanol), about which we know many things in great detail. The host (or set) is conceived as the individual organism that ingests the alcohol; as such, it is a

*Since the first draft of this chapter was submitted for publication (August 1983), methodological refinements in anthropological studies of alcohol have rendered this paragraph virtually obsolete. However, there have not been corresponding theoretical developments (cf. Heath, 1987b) that would warrant revision of the chapter, although a few references have been updated to December 1987.

complex resultant of genetic inheritance, the residua of manifold life-experiences, state of health, values, attitudes, personality, and so forth. The environment (or setting) includes not only such short-term physical factors as temperature, humidity, level of light, and so forth, but also such longer-term non-material factors as popular stereotypes of drinking and drunkenness, socially shared expectancies about how alcoholic beverages affect a person, and so forth. Most observers are quick to recognize that social and cultural perspectives are important for an understanding of the environment or setting, but it may be appropriate in this context to explain briefly why social and cultural perspectives are also indispensable to an understanding of the agent or drug, and similarly of the host or set. Epidemiologists are just as aware as anthropologists that cultural patterns influence the distribution — and sometimes even the genesis — of an agent or drug. What a people drink is more a function of the technology of brewing or distilling, agricultural practices, systems of marketing, and a variety of other customary features in that society than it is of the biochemistry or pharmacology of any beverage. While medical, biological, and psychological insights can help in understanding those aspects of the host or set that are important in affecting alcohol use and its outcomes, there are many social and cultural factors that should not be ignored. How many fatal accidents have resulted from an adolescent's view that drinking fast is a manly thing to do, combined with the expectation that a few drinks will make his date more sexually receptive, with the thrill of driving fast, and with a host of interrelated viewpoints that are commonplace in one population but unknown (or even contradicted) in another? Even if we shift from attitudes to genetics, sociocultural factors remain important; there is no human population in which couples, however much freedom they enjoy, actually mate at random. Genealogies may reflect lines of genetic descent, but they also forcefully and vividly reflect social and esthetic norms, personal tastes, arbitrary social categorization, and so forth.

The editors of this volume, in response to an earlier and more cautious draft of this chapter noted: "You are...sensitive...to issues in the philosophy of science.... The typical reader, however, will have less rigorous notions of what a theory is.... Again, we want you to highlight the underlying coherence of these models/constructs; the 'instruments' look and sound different, and are handled differently by their players, but they do combine to play a recognizable, albeit complex tune" (Chaudron, 1984). With this encouragement, and with a fresh appreciation of the fact that few other disciplines offer anything much more coherent in the way of "theories about alcoholism," I welcome the opportunity to show how anthropologists and sociologists are in the process of articulating an emergent theory of alcohol use and its outcomes.

They have done this by focusing attention on social and cultural variables that otherwise tend to be overlooked or are presumed to be more nearly uniform than is the case. In simplest terms, their approach can be phrased as follows: The drinking patterns of a given population vary as do the beliefs, attitudes, and values that members of that population hold with respect to beverage alcohol and its interaction with the human organism. At one level, such a formulation may seem so self-evident as to be banal. At another level,

however, it has been painfully obvious until recent years that many people seriously concerned with alcohol have either totally ignored beliefs, attitudes, and values or have dismissed them as being of minimal analytic and interpretive significance.

A simple, but important, corollary is: The social, psychological, economic, political, and other non-organic outcomes of drinking in a given population vary as do the beliefs, attitudes, and values that members of that population hold with respect to beverage alcohol, its interaction with the human organism, and the propriety of given behaviors.

It is apparent that the focus on social and cultural factors — shared patterns of belief and behavior — constitutes a conceptual core that is distinctive, coherent, and analytically applicable. At the same time, it has broad and immediate relevance for practical application, in terms of reducing (or increasing) the incidence of given behaviors, effecting policy, and inducing other kinds of change. With reference to alcohol use and its outcomes, this approach offers valuable opportunities for coherent description of complex situations that might not otherwise be comprehensible. But its value is not merely academic. It is also eminently practical, inasmuch as it facilitates insights about education, prevention, and treatment.

To doubt that such an important conceptual contribution approaches the elucidation of a theory probably reflects the widespread but incorrect view that any theory, in order to be plausible, must yield a set of simple propositions that can easily be tested in the laboratory by the manipulation of a few key variables. Such a view is fallacious, as is vividly illustrated in the case of geology or astronomy. Both of those disciplines have long been respected as among the most precise and predictive sciences, despite the fact that their methods are strictly observational and there is no feasible way of manipulating the key variables on which their theories are based. Similarly, to emphasize patterns of belief and behavior, especially norms and expectations shared within human populations, allows for meaningful interpretation of many events as they occur (with respect to drinking, drunkenness, drunken behavior, alcohol-related problems, etc.). This is the case not only with reference to social groups, but often with reference to individuals as well. That same analytical focus also allows for reliable predictions, effective mediation of changes, and a number of other measures that are normally accepted as appropriate tests for a theory.

Few social scientists have endeavored systematically to explore the full implications of such a sociocultural theory about alcohol. For this reason, the central idea is only casually linked to a variety of models that focus on selected aspects of it. In the present state of anthropological studies of alcohol, however, many practitioners would probably not be so comfortable attempting to trace out the lineaments of a general theory, when it is still so helpful to deal with the various models as convenient ways of looking at the relevant phenomena. Such models, or representations, make it easier to identify the many and varied parts of the behavior under study, and to understand some of the ways in which those parts relate to each other. In many instances, such models may overlap or complement various of the theories that are discussed elsewhere in this volume as deriving from the perspectives of personality, conditioning, or social

learning, or based on political, economic, or systems analysis. Most readers will presumably see this as an advantageous feature of alcohol studies, presaging greater collaboration with a promise of fuller understanding, rather than a weakness.

2.1 THE SOCIOCULTURAL MODEL

In general terms, various authors have found it helpful to refer to "the sociocultural model" to encompass the widely accepted proposition that different beliefs and attitudes about alcohol and its effects, combined with beliefs and attitudes about how, what, where, when, and with whom one should (or should not) drink, together with attitudes about the meanings of all of those, are directly related to the frequency with which problems are associated with drinking, and to differences in the nature of such problems when they occur, in various cultures.

The sociocultural model, phrased in such general terms, has come to be so widely accepted in recent years that some readers may be surprised at the breadth and specificity of the early formulation by Bales (1946). He identified three social and cultural variables that can influence the rate of alcoholism, and posited that a society's actual rate was the resultant of their interaction. First is the dynamic factor, the degree to which a culture creates inner tensions in its members. Second is the normative orientation factor — whether societal attitudes toward drinking are typically abstinent, ritual (using alcohol for sacred purposes), convivial (stressing social solidarity), or utilitarian (medical, for personal satisfaction, or symbolic as in closing a court judgment, business deal, etc.). Third among Bales's factors is the degree to which a given culture provides alternative ways of coping with psychic stress.

In that historically important paper, Bales made his point dramatically by comparing Jewish and Irish drinking patterns and their outcomes. Orthodox Jews, who had been introduced to wine as an integral sacramental part of religious ritual in the heart of the family, showed a virtual absence of alcohol-related problems, although all of them occasionally drank; by contrast, Irish Americans, forbidden to drink in their youth, were abruptly introduced to whisky in an ambience where men consider getting drunk an important part of comradeship, and often rapidly progressed to the point where they suffered major alcohol-related problems in physical and mental health.

A series of other authors have found similar interpretations to be helpful and satisfying to their own interests in accounting for the many different kinds and rates of problems that occur among human populations. Unfortunately, few such authors have been parsimonious, logically compelling, or even consistent in defining or otherwise operationalizing the variables they had in mind. For this reason, it is peculiarly difficult to characterize in summary terms this model that is probably the most widely shared among social scientists. Recent reviews of problems and prospects with relation to the sociocultural model of alcohol use provide fairly detailed critiques and comparisons with other models (see, e.g., Frankel & Whitehead, 1981; Heath, 1980a); in the present paper, my aim is more to be extensive in explication of the wide range of anthropological theories than to be intensive in analysing any individual one among them.

The general proposition that social and cultural factors affect drinking patterns has been widely accepted since the 1950s. Only after researchers were firmly convinced of that were they open to the corollary proposition that social and cultural factors also affect the ways in which people's other behavior is influenced by drinking. A pioneering study combined historical and ethnographic data from around the world to demonstrate systematically that drunken comportment is learned in ways that fit with the expectations of the population, and is subject to societal constraints, just as is the case with other forms of patterned behavior (MacAndrew & Edgerton, 1969). During the 1970s and 1980s, a rapidly expanding literature that explored "the expectancy effect" under controlled conditions in the laboratory yielded abundant and varied quantitative evidence that provides compelling support for the insightful conclusions that MacAndrew, a psychologist, and Edgerton, an anthropologist/sociologist, had inferred from the similarly abundant and varied qualitative evidence that they had analysed. Their clear and unequivocal conclusion was "that drunken comportment is an essentially *learned* affair.... *Over the course of socialization, people learn about drunkenness what their society 'knows' about drunkenness; and, accepting and acting upon the understandings thus imparted to them, they become the living confirmation of their society's teachings*" (MacAndrew & Edgerton, 1969, p.88; italics in original). In terms of the sociology of knowledge, one cannot help but wonder whether these ideas might have gained broader and more rapid acceptance if they had been presented in a scholarly journal rather than in a delightfully written little book. Much more important, of course, is the present growth of adherence to their view, even among researchers whose primary training is in the biological and medical sciences (e.g., Ewing & Rouse, 1978; Paredes et al., 1977).

Although I have discussed a general sociocultural model in terms that defy quick and easy codification, the relevant variables are real and important. There have indeed been efforts to define and operationalize more specifically the nature of those different beliefs and attitudes that are directly related to variations in the rates and types of problems that are associated with alcohol use in various populations. Most of the rest of this chapter is a catalogue of those efforts. In many instances, the names and models were not proposed by the authors cited, and I have taken the liberty of classifying and labelling them in a preliminary way that emphasizes those aspects of social life that predominate in the analysis.

2.1.1 *The Normative Model*

The beliefs and attitudes that vary with rates and types of alcohol-related problems do not, of course, comprise the total range of beliefs and attitudes that can be found within the population in question. They are more especially those that sociologists and anthropologists call norms, predominant understandings about "the rules of the game." Among the rules that are pertinent with respect to alcohol use, for example, are: who (by age, sex, marital status, and/or other socially significant criteria) may drink; when they may drink; in what manner; what associated behaviors are acceptable; and so forth. Implied in any such categorical listing are a host of parallel rules, many of which are not only logically associated but also socially pertinent, such as: who may *not*..., and so

forth; who *must* ..., and so forth. In short, the norms usually are conceived as setting guidelines — both prescriptive and proscriptive — for behavior, which the majority of people follow closely.

A frequent source of confusion with respect to the normative model is the various meanings of the word “norm,” even among sociologists and anthropologists. One usage might be characterized as “the normative,” in which a norm is regulative, what ought to be — a rule *for* behavior. Another usage might be called “the normal,” in which a norm epitomizes what members of a population consider the actual state of affairs — the quasi-statistical norm, or “average” within a population, a rule *of* behavior. Occasionally another usage occurs that might be called a derived norm, the modal or descriptive pattern — discerned by the social scientists, and not necessarily congruent with the beliefs held by members of the population in question — a rule *from* behavior. It is commonplace in culture that these kinds of norms are not necessarily the same; in fact, they often differ significantly from each other.

However imprecise it may at first appear, the normative model has continued to be the basis for some of the most explicit theorizing that social scientists have offered with respect to alcohol use. According to Ullman (1958, p.50): “In any group or society in which the drinking customs, values, and sanctions — together with the attitudes of all segments of the group or society — are well established, known to and agreed upon by all, and are consistent with the rest of the culture, the rate of alcoholism will be low.” Although Ullman’s formulation fits well with many populations, Blacker (1966) pointed out that the high rate of alcohol-related problems among the French seemed to contradict it. Noting that generality and consistency of norms may be sufficient to account for ambivalence but that the content of norms is also important, he proposed that the original formulation would be stronger if it were specified that the “drinking customs, values, and sanctions” mentioned by Ullman “are characterized by prescriptions for moderate drinking and proscriptions against excessive drinking” (Blacker, 1966, p.68).

Even though the variables are not easily quantifiable, those formulations are more comprehensive than most in the sociocultural model in terms of explicitly specifying what the variables are. This specificity allowed Whitehead and Harvey (1974) to “test” the Ullman–Blacker hypothesis against a large corpus of cross-cultural data and to find it less predictive of alcohol-related problems than the single distribution model (see below, §2.2). Nevertheless, they found the single distribution model unsatisfactory for other reasons, and resolved to strengthen the Ullman–Blacker normative formulation by inserting into it a further elaboration of the norms to include those “that keep per capita consumption low enough that few persons in that society will consume in excess of 10 cl of absolute alcohol per day” (Whitehead & Harvey, 1974, p.65).

Although the variables are not so carefully delineated, Pittman’s (1967) reformulation of the general normative model has become well known and widely accepted. He followed Bales in offering a fourfold typology of normative standards that a culture may have with respect to alcohol use: abstinent; ambivalent; permissive (toward drinking, but negative toward

drunkenness); or overpermissive (including acceptance of drunkenness and various alcohol-related problems). On the basis of a few selected cases from around the world, he posited that the permissive norm is least likely to result in alcoholism, and the ambivalent most likely. The other two norms appear, in some instances, to be associated with high rates, and in others with extremely low rates. It may seem paradoxical that individuals reared in an abstinent ambience are at high risk for alcohol-related problems, but we find everywhere that, among those who drink at all in such a normative context, many proceed rapidly to problematic drinking. Incidentally, this phenomenon fits with the suggestion by Mizruchi and Perrucci (1962) that the quality of norms is more important than their specific content, with proscriptions generally more problematic than prescriptions. The reason proposed is that once having transgressed a major proscriptive norm, one has gone "beyond the pale" and has no further guidelines for behavior, whereas anyone who violates a prescriptive norm can still relate his or her behavior to other relevant guidelines.

The six subsections that follow are brief discussions of various ways of looking at alcohol that are also normative in the sense of focusing primarily on those "rules of the game" that predominate within a given population. However, each of these — deviance, labelling, reference groups, anomie, the "time out" hypothesis, and ambivalence — has a special emphasis that distinguishes it from the other normative models.

2.1.1a Deviance. Sociologists have tended to focus more attention on what they call social deviance than have sociocultural anthropologists, perhaps because alternative institutions (such as prostitution, skid row, "the drug subculture," etc.) flourish in pluralistic urban societies where there are enough individuals who deviate in a similar manner from the dominant norms that reflect "official" morality (see, e.g., Rubington & Weinberg, 1973). In an abstaining society, however, an individual who savored an occasional beer might be viewed as intransigently deviant and hence subject to punishment, ostracism, or some other social pressure. Analysts who emphasize deviance in relation to alcohol-related problems are concerned about norms, but they focus less on the view of norms held by the actor than on the views of norms held by others who are in a position to judge the limits of acceptable behavior; that is, they focus on group norms rather than individual norms.

2.1.1b Labelling. Labelling is often a concomitant of deviance, and the labelling approach to the analysis of social behavior is a direct outgrowth of the normative model. The focus is again on the deviation of a subject's behavior from that approved by a judgmental constituency, which results in the imposition of negative sanctions. In this instance, the sanction for a disapproved act is the imposition of an enduring label of disapproval on the individual. A simple example is the label of "the drunken Indian," a stereotype widespread throughout the Americas, whereby a single act of inebriation that might well be excused or ignored in a member of another ethnic group may result in an individual's being labelled "a drunken Indian" (Westermeyer, 1974). A long-term social significance of labelling is that it may become a sort of self-fulfilling prophecy — an individual who is expected to behave like "a

drunken Indian” may find it convenient to do so, or at least not worth the trouble to try to prove otherwise.

2.1.1c Reference groups. The normative model has been applied not only to individuals, as noted above, but also to ethnic and religious segments within pluralistic societies. In effect, it pertains to situations in which different sets of norms prevail, and the norms of one reference group or “ingroup” contrast with those of an “outgroup,” which is usually dominant in political and/or economic terms. In such a situation, adherence to ingroup norms about drinking may have enormous symbolic significance in differentiating members from the rest of the population; violation of such norms is not only viewed as morally reprehensible within the group but may also put the group at risk in relation to others (who are often viewed as hostile).

Minor variations of this model have recurrently been invoked to account for the relative sobriety of Jewish populations. The philosopher Immanuel Kant (1798/1978) presaged modern sociological interpretations when he pointed out that the widespread and often virulent prejudice that Jews confront around the world had probably prompted them to be extremely circumspect about their behavior in general, and specifically to avoid intoxication because of the risk of offending people. On the basis of the most detailed study of Jewish drinking by modern sociological methods, Snyder (1958a) emphasized the point that contemporary Jews in the United States still think of drunkenness as a Gentile trait, and of their own moderate drinking as an important one among many characteristics that distinguish them (the “ingroup”) from others (the “outgroup”; Snyder, 1958b). Among the various reasons proposed to account for abstinence among Muslims is a similar emphasis on distinctive behavior as a valuable means of marking the boundaries of ethnic reference groups; similarly, Armenians (Freund, 1980), Black Hebrews (Singer, 1980), and Greeks (Blum & Blum, 1969) seem all to cherish their sobriety as an important feature that distinguishes them from less temperate populations with which they are in close and sustained contact.

2.1.1d Anomie. The concept of anomie as a kind of stress over conflicting norms has gained widespread usage in the social sciences, largely through the writings of Durkheim (1897/1951) and Merton (1957). Like the term “norm,” the term “anomie” is also used in various senses, and not all authors are careful to distinguish among them. As originally proposed, with reference to sociocultural systems that were presumed to be relatively homogeneous, anomie referred to the occasional disjunction between the norms held by an individual and those of the dominant society (somewhat akin to “deviance,” as discussed above). As the reality of normative diversity and other kinds of cultural pluralism within societies became increasingly recognized, anomie was given a new meaning—the occasional disjunction between the norms that an individual shared with the dominant society, and an individual’s opportunities to realize those norms. Members of “minority” populations can be cited as anomie, then, for very different reasons: if they hold to “minority” norms that contrast with majority norms, or even if they embrace the majority norms but are frustrated (by lack of training, prejudice, or other reasons) in their attempts to change their behavior accordingly. This latter type of anomie

has been epitomized in the popular press with allusions to “the marginal man,” rural migrants to cities who are “caught between two worlds,” and so forth.

Much of the literature that has to do with acculturation, dependency, and similar dominant/subordinate relationships among populations seems to be based on this latter model, although the terms “stress,” “strain,” “anxiety,” “acculturative pressures,” and so forth occur more often than does “anomie.” Much of this writing is based on an apparent presumption that the latter kind of anomie is a major etiological factor in the heavy drinking and/or various alcohol-related problems that occur among individuals who are in the course of attempting some significant change in their social status. Such a view dominates among recent writings about American Indians (Heath, 1983) and Eskimos (Klausner & Foulks, 1982); it was presaged by Cheinisse (1908) when he compared increasing rates of problems of rural-to-urban migrants with constant low rates among long-term urbanites, both groups being Jews in France.

2.1.1e The “time out” hypothesis. A striking insight was provided by MacAndrew and Edgerton (1969) on the basis of their review of several historical and ethnographic studies of drunken comportment. Their point is that many cultures have a certain flexibility in their norms, such that individuals are considered less responsible for their actions — and hence relatively blameless for transgressions that occur while they are intoxicated. Drunkenness provides a sort of “time out” during which many of the rules of the game are temporarily suspended. The phenomenon of occasional “time out” is a common one in worldwide perspective, and alcohol is only one of many cultural markers that are associated with it. They suggest that “the option of drunken Time Out affords people the opportunity to ‘get it out of their systems’ with a minimum of adverse consequences” (MacAndrew & Edgerton, 1969, p.169).

The authors take pains to point out that not all of the rules are suspended, however, and that people enjoying such “time out” are still subject to “a within-limits clause.” That means that disinhibition is selective — suggesting that it is a product of social learning rather than the unmediated pharmacological impact of alcohol on the central nervous system. Anthropologists have found the idea helpful in describing and analysing drinking as it relates to other behaviors in several cultures (e.g., Burns, 1980; Hill, 1978, 1980). It is used more as a descriptive than an analytic tool, but has the advantage of focusing our attention on the occurrence and nature of norm violations by intoxicated persons, which may be early predictors of alcohol-related problems in their lives.

2.1.1f Ambivalence. The works of Ullman and Blacker (cf. §2.1.1) have often been linked with an early paper by Myerson (1940), because they all include explicit reference to ambivalence as an etiologic factor in alcohol-related problems. The ambivalence to which this refers is not an individual’s liking some aspects of alcohol use and disliking others but rather a lack of consistency among the norms that a population holds about alcohol. Illustrative are the general North American taboo on drinking by children and at the same time a widely shared view that drinking not only is permissible for adults

but is a valuable skill in business and social relations. Within the same normative system, there may be strong pressure on a guest not to decline a drink offered by the host, but at the same party there is likely also to be embarrassment or disgust if a guest becomes offensive while drunk.

The qualities of consistency and consensus have long been recognized as crucial with respect to the strength of norms as regulators of social behavior; those same qualities are obviously also important in terms of the mental health of individuals who can easily be confused or frustrated by the mixed signals that come from inconsistent or unpopular norms. Myerson's essay briefly reviewed the conflict between hedonism and asceticism in the history of Western culture, and attributed alcoholism (in the sense of problems stemming from alcohol use) to this societal ambivalence. Several other authors have pointed to contemporary inconsistencies in norms—for example, the North American taboo on drinking by children, coupled with the view that hard drinking is a sign of manhood, that cocktail parties are sophisticated, and so forth—as examples of ambivalence that put individuals at high risk with respect to alcohol use. This literature has already been reviewed in considerable detail by Room (1976), who effectively makes the point that it is of dubious utility; however logical allusions to ambivalence as an etiological factor may be, inadequate definitions and lack of precision in dealing with what is basically a state of mind posited in others are problematic.

2.1.2 *Culture-Specific Models*

In a wide-ranging discussion around the theme of theories on alcoholism, there is probably little to be gained by cataloguing the diverse psychological and sociocultural factors that have been invoked by one or more social scientists to account for the ways in which various peoples drink, or for the ways in which drinking affects the members of those populations. Such studies usually show minimal familiarity with the ethnographic, sociological, or other theoretic literature on alcohol use, and make no attempt to compare the local ambience with any other. Because this kind of modelling is intentionally culture-specific (sometimes including a disclaimer against any intention that the interpretation be applied in any other context), I mention it only as a genre that occurs in some ethnographic reports but has little relevance to the emergence of an anthropological theory of alcohol use and its outcomes.

A few examples illustrate that this type of treatment of drinking patterns, while useful for microscopic description and analysis of particular populations, adds little in relation to more broadly conceived theories and models. One author links the curative value that a Roman Catholic vow holds for members of one North American Indian tribe to the belief that drunkenness is a kind of demonic spirit possession (Stevens, 1981). Another sees the justification of abstinence by conversion to Protestantism as a way in which ambitious Mexican peasants can avoid spending on expensive community fiestas (Kearney, 1970). An African tribe's profits from moonshining are said to be sufficient motive for them to stay sober while encouraging neighboring populations to have more and longer binges (Omori, 1978). All these models have in common the view that intoxication or sobriety is obtained because of functions to the behaving person.

2.2 THE SINGLE DISTRIBUTION MODEL

Another causal relationship between the rate of occurrence of alcohol-related problems in populations and social and cultural factors has been alternatively called “the single distribution model” or “the distribution of consumption model.” These names both relate to key variables in relation to per capita consumption of alcoholic beverages, which a French statistician/sociologist (Ledermann, 1956) suggested is crucial in estimating the prevalence of alcoholism. He asserted that the distribution of consumption of absolute alcohol within any given population follows a markedly one-sided lognormal curve. The aspect of “single distribution” relates to the fact that, in such a figure, the heaviest drinkers lie along the extremely gradual slope of the “long tail” of the curve, in contrast with some other postulated figures in which the heaviest drinkers appear to be discontinuous with the rest of the population. For example, if alcohol consumption followed a bimodal curve, the heaviest drinkers would appear anomalous, as the minor “blip,” markedly set off from most of the drinking population. An extremely valuable contribution of the single distribution model is recognition that the dividing line between “heavy” or “excessive” drinkers and others is arbitrary, and that there is no gulf separating their consumption from that of some others who are not so labelled.

The single distribution model has gained considerable popularity since the 1960s, especially among those who find reassurance in quantification. On the basis of empirical studies with small samples within a few international populations, Ledermann (1956) arrived at the conclusion that the consumption of alcohol among individual members of any homogeneous society follows a lognormal distribution. With the recognition that a certain percentage of the population abstain, and that it is practically impossible to consume more than 100 cL of absolute alcohol daily, the curve can be drawn on the basis of “apparent consumption,” which can be derived from records or receipts for liquor taxes, records of sales of beverages, or other sources that are often available in modern societies.

Given the data on consumption and the assumption of lognormality, and the assumption that cirrhosis varies directly in proportion to consumption, the distribution of consumption is thought by some to provide a relatively simple quantitative basis for estimating rates of alcoholism.

The link between cirrhosis death and problem drinking has been the subject of an enormous literature in the field of epidemiology, research methods, and theory ever since it was tentatively proposed as a heuristic device by Jellinek (1951). After several attempts to refine “the Jellinek formula,” its author abandoned it as misleading; a variety of reasons were involved, but one of the principal ones was the diversity of non-physiological problems that are important in what Jellinek himself had come to refer to as “species of alcoholism” or “the alcoholisms” (Jellinek, 1960). Nevertheless, researchers from various countries have continued to experiment with ways of calculating what they hope might be a realistic response to the commonplace question, “How many alcoholics are there in such-and-such population?”

Few proponents of the single distribution model would phrase it in such terms, but the single distribution model can well be viewed as a variant of the

sociocultural model, with the mean consumption tied to the nature of societal norms. It is appropriate, albeit ironic, that Ledermann early made the point that his statistical approximations would apply only within culturally homogeneous populations (Ledermann, 1956), a limitation that has generally been ignored by recent spokespersons for that view.

The irony lies in two disparate facts. The first is that, since the 1970s, there has been a strong tendency on the part of a few authors to cast "the sociocultural model" and "the single distribution model" as not only contrasting but even conflicting viewpoints (see, e.g., Frankel & Whitehead, 1981; Moore & Gerstein, 1981; Parker & Harman, 1978; Whitehead & Harvey, 1974). The opposition has some relevance when the models are discussed in the limited sense of providing logical guidelines for prevention. In terms of this practical application, the sociocultural model stresses a long-term educational approach, in the expectation that altered social norms seem most likely to be a lasting basis for diminishing alcohol-related problems; adherents to the single distribution model, by contrast, emphasize direct steps to limit access to alcoholic beverages, with increased prices, heavier taxation, shorter sales hours, and so forth, with the expected diminution in consumption likely to result in diminished problems. This is not an appropriate context in which to recapitulate this controversy, but it does deserve mention that, as theoretical approaches to the understanding of how and why people drink as they do, and have (or do not have) various problems as a result of their drinking, they are complementary rather than conflicting. This point has probably been most forcefully made (by Frankel & Whitehead, 1981) in a systematic comparison of those models as predictors of the occurrence of various kinds of alcohol-related problems in various cultures around the world.

Another parallel between the single distribution model and the sociocultural model makes their supposed opposition ironic, and that is their shared emphasis on normative aspects of behavior. When Ledermann spoke of "the snowball effect" to characterize the ways in which individual decisions about drinking reflect the perceptions of how others drink, he described it in terms that are very similar to what social scientists have more recently been referring to as "modelling," "socialization," "peer pressure," "adherence to norms," and so forth.

The single distribution model has enjoyed increasing cachet in recent years as it has been emphasized by various agencies concerned with public health (cf. §5.2). In each of these instances, however, there has been a generalization beyond Ledermann's original formulation (which emphasized the link with cirrhosis) to a posited correlation with the broad range of drinking problems in general.

2.3 THE ANXIETY MODEL

One of the earliest anthropological models relating to alcohol use is still one of the most widely quoted and influential among all of the "theories on alcoholism." Horton's succinct formulation remains forceful 45 years later: "*the primary function of alcoholic beverages in all societies is the reduction of anxiety*" (Horton, 1943, p.223; italicized in original). This was the first

hologicistic (then called "cross-cultural") study in which the associations between psychological factors and overt patterns of behavior were tested, using a worldwide sample of cultures as cases, relying on extant ethnographic reports as sources of data. Methodological details will be summarized below (§3.2), but it seems appropriate to mention here that the nature of the sources required that Horton devise selected indices of the generalized "anxiety" factor; he chose fear of food shortages, sorcery, and acculturative pressures as bases for anxiety that were generally well reported in the literature. Despite the imprecision of his measures, Horton strove to offer a model for a new kind of scientific research, and the impressive successes achieved within a couple of academic generations attest to the value of that approach (Levinson, 1977). In his concern for conformity to scientific criteria, Horton even spelled out a couple of "theorems": "The strength of the drinking response in any society tends to vary directly with the level of anxiety in that society," and, as a corollary, "to vary inversely with the strength of counteranxiety" (Horton, 1943, p.293). Statistically significant support was found for both propositions among the sample of 56 societies about which there was sufficient information at that time.

Several authors have subsequently emphasized anxiety, stress, tension, and related individual concerns as major etiological factors in problem drinking, not only with reference to individuals but also with respect to differential rates among populations (Schaefer, 1976).

A particular kind of anxiety that is often cited with respect to alcohol-related problems is that which accompanies sociocultural deprivation — the alienation of a population from the norms and other aspects of culture that had been meaningful and valuable to them. The phenomena of sociocultural deprivation and anomic depression relate to the discrediting of socially held norms, but the emphasis is not on norms about alcohol so much as on more fundamental ideas about what is good and right. Those values are still cherished by the individuals concerned; the individuals suffer such deprivation or depression because their values have been brought into question by members of another society, typically one that is politically and/or economically dominant. In a fundamental way, this kind of confrontation does more than pose serious choices for members of the dominated population; it may be gravely disorienting inasmuch as it undermines traditional knowledge and authority, implies that old views (e.g., of the supernatural) may no longer be relevant, and erodes one's concept of self and one's relation to the rest of the world.

Sociocultural deprivation is one kind of anomie, as briefly mentioned above (see §2.1.1d); the generic term is often used, without any qualifier, in formulations that are intended to account for the occurrence of alcohol-related problems in certain individuals or to account for a high rate of such problems among a population. Our usage here accords with that clearly delineated by Jilek (1981) as "anomic depression," emphasizing not only the absence of an effective normative structure but also cultural confusion and relative deprivation. Related to sociocultural deprivation is the emphasis on "stake in society," proposed by Honigmann and Honigmann (1970) and applied in some detail to

the case of the Navaho Indians by Ferguson (1976).

An anthropologist who was also a North American Indian pointed to the historical and progressive pressures of sociocultural deprivation that have been suffered by his fellows and suggested that their heavy use of alcohol was largely an attempt to relieve "the Indians' deep sense of inadequacy and inferiority" (Dozier, 1966, p.77). Similarly, a psychiatrist working with various non-Western populations found their different rates of alcoholism "at variance with psychoanalytic theory [but] sociocultural deprivation which affects self-image and creates ego-need and inferiority feelings seems a likely explanation" (Beaubrun, 1967, p.655).

2.4 THE SOCIAL ORGANIZATION MODEL

The method of holoeistic research that was pioneered by Horton (1943), in his testing of the anxiety model, has been appreciably refined by other scholars who have conducted large-scale correlational studies to test the covariance of various traits with institutions, using a worldwide sample of societies as their subjects. One of the early efforts used the same sample that Horton had used; similarly, it focused not on alcohol-related problems so much as on drinking behavior. Instead of devising indirect indices for psychological states, however, Field (1962) selected elements of social structure and social organization that are more clearly discernible and institutionalized. His findings never caught the imagination of the public, nor did they have much impact among scholars even though they were more statistically significant, in many instances, than Horton's had been. Who but a social scientist could be moved by the realization that the relatively rare phenomenon of communal sobriety is linked with patrilocal residence, bride-price, village settlement patterns, and lineages, and that "drunkenness in primitive societies is determined less by the level of fear in a society than by the absence of corporate kin groups with stability, permanence, formal structure, and well defined functions" (Field, 1962, p.58)?

2.5 THE CONFLICT-OVER-DEPENDENCY MODEL

As the method of large-scale cross-cultural studies rapidly gained acceptance, it was used to examine the interrelations of aspects of child training on the one hand, and the dominant institutions of cultures on the other.

An ambitious effort to ascertain whether factors in socialization and informal education might be systematically linked with patterns of drinking and drunkenness was undertaken jointly by psychologists and an anthropologist (Child et al., 1965). Unfortunately, many who discuss their work appear not to have read it carefully, and confuse the sociocultural "conflict-over-dependency" model with psychological or psychoanalytic "dependency" model (cf. Barry, Chapter 4 in this volume). Cross-cultural comparisons of child training often show marked discontinuity in relation to the roles expected of adults. When indulgence of children is normal and is followed by heavy demands for self-reliance in adults, Child et al. suggest that the society's members are at high risk for conflict over dependency. Such individuals often find that "as a reaction to dependency conflict, alcohol has a triple function: it reduces

anxiety and tension; it permits the satisfaction of desires for dependence; it permits uncritical indulgence of unrealistic fantasies of achievement" (1965, p.31).

2.6 THE POWER MODEL

Both the psychoanalytic dependency model and the conflict-over-dependency model are often discussed as if they stood in marked contrast to the power model, although those scholars who are most familiar with them tend to stress complementarity rather than contrast. A series of essays by several psychologists were compiled and revised in a book that emphasizes that "Men drink primarily to feel stronger. Those for whom personalized power is a particular concern drink more heavily. Ingestion of alcohol cues thoughts of strength and power in men everywhere, apparently for physiological reasons" (McClelland et al., 1972, p.334). Although this view can be construed as primarily psychological in its theoretic bases and relevance, it deserves to be mentioned among anthropological and sociological approaches. One reason is the explicitly pan-human scope of the formulation — many other psychological theories may be intended to be cross-culturally applicable, but few make the point emphatically as these authors do. Although this theory did not emerge from a holocultural study, it was strongly supported by one. The authors' confidence that the power model is not ethnocentric derives, in large part, from the fact that they tested it in an imaginative way by studying the linkages between content analysis of folktales (for themes of power) and prevalence of drinking, in a large sample of societies around the world. They found close association between themes of inability, weakness, and dependence on the one hand, and heavy drinking on the other, which they interpreted as insecure individuals' having recourse to alcohol for the feeling of magical potency it conveys. Proponents of both the power model and the conflict-over-dependency model have agreed that they are, in essence, dealing with two sides of the same coin in terms of psychosocial dynamics, with those who suffer conflict over dependency being likely to want to derive feelings of power from drinking.

2.7 THE SYMBOLIC INTERACTIONIST MODEL

Since the late 1950s there has been increasing dissatisfaction on the part of some social scientists who consider that normative interpretations of culture seem too narrowly deterministic and make little allowance for individual variation, including innovation, deviance, and similar patterns of behavior that do not fit the norms. A small but articulate group have caught the imagination of laypersons as well as scientific colleagues by discussing behavior patterns in dramaturgical terms, treating behavior as roles (in the theatrical and not the sociological sense), discussing private affairs as "backstage," and so forth (e.g., Goffman, 1963). Illustrative of this symbolic interactionist model, as it relates to drinking, are Lurie's (1971) characterization of North American Indian alcohol use as "the world's oldest on-going protest demonstration" or Braroe's (1975) sensitive insights into the manipulative value that "the drunken Indian" stereotype affords in dealing with whites in a Canadian town.

2.8 SOCIALIZATION AND SOCIAL LEARNING AS MODELS

The behavioral malleability of the human neonate is dramatic evidence that social learning is the cornerstone of culture. Not only is the content of social learning a function of culture — what a people know, what they consider important, what they believe they cannot know, and so forth — but so is the process (who teaches what, in what manner, with what kinds of rewards and punishments, toward what ends, and so forth).

Because conditioning and social learning theories are discussed in considerable detail in other chapters of this book, it would be redundant to elaborate on them here. For those who may not be familiar with the scope of the social and behavioral sciences, however, it may deserve mention that anthropologists and sociologists have both learned from and contributed to the literature on those subjects.

2.9 FUNCTIONAL INTERPRETATIONS AS MODELS

Among the few controversies that have had any broad impact among social scientists during the past few decades has been increasing dissatisfaction with the once-predominant view that cultural features can be "explained" by outlining the functions that they serve — for an individual, for the social system, or for the culture itself. Critics hold that such a view presupposes a degree of logical consistency and integration that is the exception rather than the rule; that strict adherence to functionalism would deny the possibility of deviance, cultural change, and many other other dis-integrated features that we know do characterize most sociocultural systems most of the time.

Despite these shortcomings, the logic of functional analysis, linked to concepts of adaptation and adjustment of individuals and of populations, still holds considerable appeal. In fact, a major portion of the ethnographic literature on alcohol use and its outcomes is informed by little more in the way of theory than a concern with discerning what benefits (functions) and what costs (or dysfunctions) accrue that might account for the occurrence (and presumed continuity) of the patterns described. However, although post hoc functional interpretations of alcohol use and abuse in a culture are frequently made by sociologists and anthropologists, as such they do not constitute models.

3. CHARACTERISTIC RESEARCH METHODS

It may seem curious that, after having indicated that there is no anthropological theory on alcoholism, I have summarily discussed so many models that are relevant, and have briefly alluded to some others that can be found in the literature but that have gained little currency. Beyond the sheer number and variety of models based on social and cultural factors, it is also striking that so many of them have gained popularity and acceptance among laypersons and scientists alike, and are widely applied (usually without attribution) in discussions of alcohol-related problems. These factors are all the more remarkable in view of the fact that most of the models have been applied *post facto*; they

epitomize the inductive approach, in which data are collected on a subject without being shaped by the prior imposition of preconceptions.

In such a situation, some might consider it anomalous to devote much attention to the methods that characterize sociological and anthropological research on alcohol. On the contrary, such an effort has special significance in this context because it sheds unusual light on the scientific enterprise, revealing some of the ways in which data and theory are related to each other. It also shows how methods that many consider pedestrian and of limited value can, when carefully applied, yield significant insight that could not be gained by more controlled methods of research.

This discussion of characteristic research methods does not replicate the organization of the preceding section, for various reasons. The research methods themselves comprise the organizing principle for this section, with mention of the models or theories sometimes of considerably less importance than is the case in other chapters that deal with more deductive approaches, where methods are devised with the aim of carefully and narrowly testing specific hypotheses or theories.

3.1 ETHNOGRAPHIC METHODS

In one sense, none of the various anthropological or sociological models of alcohol use derives in a clear and direct sense from ethnographic methods, nor have such methods been used in a clear and direct manner to test them. In another sense, however, most of the models discussed above derive indirectly from them, in that a major portion of the data were collected and reported that way. This apparent paradox is another outcome of the unusual but fundamentally important fact that most sociocultural formulations about alcohol use and its outcomes have been serendipitous by-products of research efforts that had very different or quite unrelated aims.

The primary task of ethnography is to provide a detailed description of a particular corpus of human behavior; to report "what's going on" in such a way that the description can serve as data for subsequent use by various persons who may bring a wide range of analytic and interpretive skills to bear. This is not an appropriate context in which to recapitulate the philosophic — and sometimes heated — debate that has occurred within the social and behavioral sciences, especially during the 1960s and '70s, over "objectivity," "selectivity," "positivism," "phenomenology," and related issues. There is a growing minority among anthropologists who have little patience with what they call "mushy data," or with traditional methods that they consider "journalistic." It is ironic that this development within the discipline coincided with a contrasting movement in many other broad fields of study (notably education, health, and other) in which ethnographic methods were gaining recognition as useful tools for collecting data and arriving at insights that would not be accessible by other means (Akins & Beschner, 1980; Heath, 1980b).

Although there are several articles and book chapters that ostensibly review anthropological research methods, most of them are written in such a way that they presuppose too much and have little value as introductions to how to do it. A welcome recent addition that fills the gap is Agar's (1980)

meticulous handbook; apart from its value as the best introduction to ethnographic research methods in general, this volume has special relevance because many of the illustrative examples derive from Agar's long-term research among drug users. The irreducible basis of ethnographic methods is the observer as the principal, if not the only, instrument of observation, measurement, and documentation. Beyond that, a serious effort is usually made to describe behavior in terms that include (but are not restricted to) the point of view of the subjects themselves. Although it is patent that no human being can come close to comprehending the vast array of variables that might influence any given act, an ethnographer strives to be as inclusive ("holistic") as possible with respect to discerning attitudes, values, and other "subjective" aspects that might plausibly exert significant influence on the actor, as well as economic, political, and other institutional and social-relational aspects of the context that might be similarly important in understanding what is happening.

Ethnographic methods yielded the vast majority of the case studies (whether of individuals or groups) on the basis of which investigators proposed theories, hypotheses, theorems, or other kinds of models that somehow "made sense" of the data. As indicated above, most of the sociocultural models of alcohol use were developed post facto, not at an early stage of planning so that they shaped guidelines for research, nor, for that matter, even during the active phase of data collection. Even more unusual is the fact that such models were often developed by persons other than those who had collected the data in the first place and had direct familiarity with the drinkers and their context.

In one sense, this pattern can be viewed as a strength of the sociocultural literature. It suggests that there is less likelihood of bias in reporting alcohol-related behavior because that was not the interest of the initial investigation. It also reflects the thoroughness of field workers who reported behavior in sufficient detail that others could put their data to unforeseen uses. In another sense, there is a corresponding limitation in the sociocultural literature. Many of the details that might interest a researcher who is knowledgeable in alcohol studies (e.g., absolute alcohol content of beverages, quantitative measures of consumption in brief periods) are too often overlooked or, at best, are reported in ways that make interpretation or comparison difficult. Perhaps the most remarkable fact about the interrelation of ethnographic methods and theories about alcoholism is the enormous impact that the products of those methods have had, even among colleagues who are unfamiliar with them.

3.1.1 *Direct and Participant Observation*

One of the key features in ethnographic research is observation, intense and sustained focusing of attention by the researcher, in an effort to perceive and record the diverse factors, both gross and subtle, that may be relevant to the understanding of ongoing human behavior. The senses of the observer may be supplemented by photography, recording (video or audio), and some other tools, but careful scrutiny and analysis of what is observed is the critical process.

There are some who would view such an approach as "unscientific" or "not sufficiently rigorous," because it does not provide for the manipulation of variables, strict control of setting, and other characteristics that are thought by

some to be distinguishing features of the scientific enterprise. However, that view ignores the remarkably precise predictions that were achieved by astronomers who relied for centuries on observational studies. Similarly, the progressive refinement of our understanding of evolution and speciation in biology, the entire corpus of vulcanology and most other fields in geology, our increasing sophistication about animal behavior in ethology, are all examples of science as primarily an observational undertaking, with few opportunities for planned experimentation.

Other critics sometimes express concern over the variability that is inherent in reliance on human beings as the principal instrument. In fact, controversies and contradictions over data are the exception rather than the rule in anthropology, although such rare exceptions often receive disproportionate publicity. There tends to be a remarkable degree of consensus among ethnographers who have worked in the same setting. Apart from that, and with specific reference to drinking, it is noteworthy that non-professional observers, after very brief training, have proven to be highly reliable (i.e., $r > .90$) in their recording of data in a naturalistic setting (Billings et al., 1976; Kessler & Gomberg, 1974).

Direct observation—that is, observation in which the researcher's preoccupation is to observe and record—is listed here as a subtype, to distinguish it from participant observation—that is, observation that occurs unobtrusively while the researcher is engaged in activities that make other kinds of sense to the people involved. This is not to imply that participant observation is surreptitious or that direct observation is not. It may be well known that a participant observer is not merely interested in what occurs but also expected to write about it later, whereas a direct observer may remain hidden behind a one-way mirror, recording behavior that the actors think is unobserved.

Participant observation is often considered the methodological cornerstone of ethnographic research, not only allowing the investigator to enjoy a helpful degree of rapport but also providing close access to a myriad of cues that might otherwise be missed. Part of the romantic mystique that some attribute to anthropology relates to a researcher's achieving empathy with the people being studied; although that does occasionally happen, it is by no means a requisite to understanding what they believe and feel, which is important but can be learned through observation and interviewing. The special value of close and sustained interaction with the people being studied lies in the appreciation it gives of the complex web of personal, political, economic, ideological, material, and other socioculturally relevant factors that comprise the context for any behavior. There have already been a couple of articles that summarize the relevance of observational methods with specific reference to the field of alcohol studies (Heath, 1981; Room, 1981); a recent volume on "social settings" comprises a collection of papers that emphasizes observational studies of drinking (Harford & Gaines, 1981; see also Single & Storm, 1985).

A major strength of observational studies lies in the fact that actual (rather than reported) patterns of behavior are accessible: that is, what people

do (which is often very different from what they say they do). Furthermore, observational studies have the advantage of providing information about some kinds of behavior that people might prefer not to talk about, and even kinds of behavior that people themselves may not be aware of. In this last category, the predominant pattern of synchronous sipping in the U.S., a remarkable example of micro-level modelling in pairs or small groups, was unrecognized even by researchers until it emerged recurrently in intensive observational studies in recent years (Billings et al., 1978).

3.1.2 *Interviewing*

However valuable observation (direct and/or participant) may be in providing insights into who drinks, when, where, how, and with what results, it can overlook a number of factors that are crucial to understanding those behaviors, their meanings, and their impacts on the lives not only of the people observed but of others as well. Few anthropologists or sociologists would be content to deal with any system of behavior without also making a serious effort to understand the values, attitudes, and feelings that are expressed in and associated with that behavior. When dealing with contemporary human populations, one of the most effective ways of doing this is interviewing.

Unlike observation, interviewing has long been the subject of intense analysis by practitioners of various disciplines, and there is an abundant literature on how to do it for various purposes. I will not here describe the details of interviewing as a research method, but it should be obvious that, when one attempts to learn about the feelings and meanings that people attach to things, respondents should at least be given some opportunity for open-ended (or unstructured) comments. I do not mean to deprecate the potential value of directed (or structured) interviews or schedules, which guide the respondent along selected channels in order to assure that key points of interest to the researcher are addressed. In many contexts, and for many purposes, such interviews are not only efficient but requisite. In most behavioral research, both approaches should probably be used, and the quality as well as the quantity of data from each would probably be strengthened by complementary data from the other. A serious danger, however, is that a misplaced concern with rigor or quantification may lead investigators to omit open-ended interviews, which often reveal factors that are all the more important because they were unforeseen.

An important point to make in this context is that, like observation, interviewing cannot be strictly cited as a research method that was specifically instrumental in the development of any of the sociocultural models of alcohol use, and yet it was basic to virtually all of them. The reason is the same as it was for observation: the serendipitous quality of much of the sociocultural analysis of drinking, deriving from ideas and questions that postdate the collection of the original data.

3.1.3 *Interpretations of Behavior*

If most ethnographic data are not collected in relation to specific theories or models, one may well wonder how the sociological and anthropological endeavors ever progress beyond mere antiquarianism, the amassing and

compiling of voluminous disconnected anecdotes. Rightly or wrongly, it is typical that the kind of interpretive analysis that has resulted in the formulation of sociocultural models of alcohol use has been retrospective, in the sense that it took place after data collection had been completed. There are a few instances in which researchers have been able to return to the field for additional work, but such an opportunity usually occurs only after the lapse of some years. In such cases, it is common to find that significant changes have occurred, so that the restudy becomes a way of discerning cultural dynamics with the earlier work as a baseline, instead of serving as a retest or replication of the original study. This genre of research often reveals important psychosocial insights — but it does little to counter the nonscientific image that some critics hold with respect to interpretive analysis.

Similarly, ethnographic case studies that are cited to illustrate anomie, rural-to-urban migration, prohibition imposed on minority populations, or other sociocultural phenomena are almost never based on the observations that were made in the field, nor even on the “raw” notes recorded by the original observer, nor on interviews as they occurred, nor even on transcriptions of them made by the interviewer. The data offered in analytic and interpretive studies are several times removed from the original data. The “original data” consist of events in which particular people say and do particular things in particular ways. Those events are ephemeral and will never recur in detail, but a keen observer can discern many components of them and, if also a conscientious and thorough reporter, may record a major portion of those observations in a way that is sufficiently descriptive and accurate so that some key elements of the original event may be evoked. It is usually at least a year later, and often in a setting far removed from the event, that a researcher selectively extracts or abstracts portions of such field notes as the substantive segment of an analytic essay, which may, in turn, be cited by another researcher who has no familiarity with the original data, nor even with the field notes, but only with the published synthesis and analysis, in which the dynamics of the event are, at best, foreshortened and the context is vastly reduced. The emphasis is usually on patterns — recurrent associations, systemic linkages, significant regularities, and so forth — as they can be discerned in data that are far removed from the original events.

It would be naive and misleading to suggest, as once was common, that patterns somehow “emerge” from the data. Nevertheless, the power of social constructions of reality is such that most expert observers can be consistent in their identification and interpretation of the psychosocial dynamics and other variables that are relevant to a given sequence of behavior. This process involves what one of the most insightful analysts of our own society speaks of as “the sociological imagination” (Mills, 1959). An equally masterful interpreter of Indic, Malaysian, and Muslim institutions likens the interpretation of cultures to literary criticism, stressing the search for meanings (Geertz, 1973, 1983). Such insights are not mystical by any means, although the analytic process may seem obscure to those who are not familiar with it. Few authors are explicit about the manifold sources of cultural norms, and many presuppose that all readers share their assumption that such norms are generally transmitted

across generations by a long and complex process of reward and punishment — such propositions are fundamental to sociocultural analysis, and yet they are seldom articulated in the context of specialized studies that have to do with alcohol use. Even when an occasional author takes great pains to spell out the conceptual scheme on which a study is based, that portion of the work is often virtually ignored; a striking example is Horton's (1943) use of "subsistence insecurity," "frequency of warfare," "sorcery," "premarital sexual restraints," and two types of "acculturation" as indices of a generalized level of societal anxiety. Critics who point out that drinking in a given society does not correlate with one or another of those indicators miss the point of his imaginative effort to measure indirectly "anxiety," which is rarely mentioned and virtually never quantified in ethnographic reports.

The process of interpretation of behavior would seem much less mystical and more convincing if ethnographers were more consistent in adhering to the guidelines that Agar offers for "narrowing the focus" (1980, pp.119-136), a step-wise procedure that combines: sampling individuals within the population (with explicit evaluation of possible biasing factors), sampling events, calculating a distribution, and providing measures of both central tendency and dispersion.

Although such interpretations of behavior may, at first, seem inappropriate or insignificant as research methods when one is attempting to understand alcohol use and its outcomes, they are fundamental and virtually pervasive with respect to contributions of the social sciences. This method is the basis of most of our understanding of the sociocultural and normative models (§2.1, 2.1.1) and their variants (deviance, labelling, reference group, anomie, "time out," and ambivalence: §2.1.1a-f), as well as the various culture-specific models (§2.1.2). Although the single distribution model (§2.2) relies heavily on quantitative data and mathematical analysis, it too is firmly grounded in assumptions that relate to such interpretations of behavior. In a similar manner, this kind of interpretation of behavior is often invoked in support of various models that have been elucidated by the more rigorous holistic method (§3.2), such as anxiety, social organization, conflict-over-dependency, or power (§2.3-2.6 respectively). This methodological step is almost certainly the most important with respect to our understandings of alcohol-related behavior in terms of symbolic interactionism (§2.7) and functional interpretations (§2.9); it probably shares primary utility with thematic analysis (§3.3) and with respect to studies of social learning about alcohol (§2.8).

Although this method of research does not conform strictly to popular views of the scientific method, the outcomes of such interpretations of behavior command both interest and considerable respect among scientifically oriented colleagues in many fields. Just as Heath's (1958) account of Camba drinking is still often cited as evidence that widespread and frequent drunkenness does not necessarily result in problems, Lurie's (1971) characterization of North American Indian drinking as a politically significant pattern is widely accepted and cited by others as well as social scientists. The fact that such an interpretive enterprise does not always yield quantitative data or even concise testable hypotheses does not mean that it is frivolous. In realistic terms,

this is the kind of information on which most of us base most of our own judgments and actions most of the time, and experience usually confirms not only the reliability but even the validity (at least, for all practical purposes) of our interpretations of behavior.

3.2 HOLOGEISTIC ANALYSIS

Hologeistic analysis is one of the few distinctive methods of modern research that is firmly rooted in anthropology; it is also one of the few distinctive methods of modern research that got significant impetus from its early role in the study of alcohol-related behaviors. Although Horton's (1943) formulation of "the anxiety model" of drinking is well known, less widely recognized is that it was the product of a novel set of procedures that have quickly evolved into a vital and influential subdiscipline within anthropology and sociology.

The hologeistic approach, often called "the cross-cultural method," attempts to arrive at broadly applicable principles, generalizations, or laws of human behavior by means of large-scale comparison among populations that represent various periods of history and diverse geographical and cultural areas. Particular institutions or aspects of culture are defined, scaled, and then measured in terms of the degree to which they co-occur with other institutions or aspects of culture, in worldwide perspective. In devising the complex new methodology for his hologeistic cross-cultural study that resulted in the anxiety model, Horton deliberately set out to define terms and to operationalize variables in such a way that measures of psychological factors (such as anxiety) could be gleaned from ethnographic data collected by observers and interviewers who had no training (nor, in many instances, even interest or awareness) with respect to the psychology of Horton's time.

The vast corpus of ethnographic literature comprises the database for this kind of analysis. Samples among relatively well documented cultures are drawn in such a way as to include various races of humankind, in different kinds of physical environments, with various kinds of social and political organization, as well as diverse religions, ecological adaptations, and so forth. One of the aspects of sampling that has been an enduring preoccupation is the attempt to avoid unwitting inclusion of cultures that are historically derived from, or in close and sustained contact with, each other, because the widespread phenomenon of cultural diffusion might confound other assumptions about the independence and covariance of selected aspects of culture.

Although this research method is used by relatively few social scientists, it has been employed both to generate and to test a significant number and variety of the models of alcohol use that have come from the social sciences. A classic example is the anxiety model, stemming from Horton's (1943) correlation of various indices of anxiety with frequency and intensity of drunkenness (cf. §2.3). The same method and the same data impressed Field (1962) with a very different set of cultural linkages that he articulated as the social organization model (cf. §2.4). Considerably more data and more conscientious efforts at sampling, with independent raters as a check on reliability of scaling variables, and other refinements were brought to bear by Child et al. (1965), still using

essentially the same methodology. Their interest in child training and its relation to adult institutions formed the basis for the conflict-over-dependency model (cf. §2.5). With less attention to sampling, and a narrow focus on psychological themes in folklore, McClelland and his colleagues (1972) adapted the hologeistic method in support of the power model (cf. §2.6).

Hogeistic research provides a striking example of the extent to which quantification has been assigned a crucial role as the distinguishing feature of "hard science" in contemporary Western culture. A remarkable amount of attention has been paid to sampling procedures and to statistical manipulations in this kind of large-scale cross-cultural research, although the "original data" at that level of analysis comprise extracts from ethnographic reports such as we have demonstrated are already far removed from the workaday realities of the behavior being studied (cf. §3.1.3). It is probably salutary that those who most use the hologeistic method are not only aware of, but also deeply concerned about, some of its apparent limitations (e.g., Naroll, 1962; Schaefer, 1974).

3.3 THEMATIC AND CONTENT ANALYSIS

Thematic analysis is similar to what I have called "interpretations of behavior" (§3.1.3), except that the latter has a definite grounding in specific actions and interactions of specific individuals, whereas the former is usually derived from any of a wide variety of literary, historical, and other sources, which often treat human behavior at a much higher level of generality. Dominant, pervasive, typical, or otherwise important themes are derived from close analysis of patterns of belief and behavior that are portrayed in sources such as folktales, films, novels, histories, travelogues, and so forth (e.g., Krippendorf, 1980). Bales's (1946) original impressionistic account of Irish and Jewish drinking exemplifies analysis that derives themes from sources that generalize about patterns of behavior; Stiver's original study of Irish-American alcohol-related stereotypes (1976) similarly typifies thematic analysis, whereas his account of recent changes (1978) appears to be more substantive an interpretation of behavior.

Just as they have been used as bases for interpreting behavior as observed in individuals and groups, the several variants of the sociocultural model have also been used as bases for interpreting and analysing themes that are extracted from diverse sources; this is the case for the normative, deviance, labelling, reference group, anomie, and ambivalence models. This is also the case for the anxiety model, including sociocultural deprivation, portions of the power model, and symbolic interactionism. It probably predominates with respect to most of what has been proposed in the way of both socialization and functional interpretations. The method may not be precise, but it is remarkably reliable, and it seems to yield insights that even the most rigorous scientist finds congenial and constructive.

In studies of literature, linguistics, semiotics, and communication, detailed methods of content analysis have been devised that have demonstrated their reliability and worth in extracting meanings and values — often implicit — from close scrutiny of texts (Stone et al., 1966). Although it would appear logical that practitioners of what I have called thematic analysis would

try to profit from the experience of content analysis, there has been little cross-fertilization, at least as evidenced in the anthropological and sociological literature on alcohol. A few studies that deal with advertising and the image of alcohol that occurs in television or in films (e.g., Cook & Lewington, 1979; Defoe et al., 1983; Partanen, 1980) are more quantitatively oriented than most thematic analyses, but they do not approach the sophistication of many content analyses.

4. HISTORICAL DEVELOPMENT OF THE EMERGING THEORY AND MODELS

In the absence of clear consensus about a dominant unitary theory, the most fruitful approach to tracing historical developments appears to be a quick overview in which the several models are put in a roughly chronological sequence, and a few of the milestones in the literature are noted, as they relate to the emerging theory sketched above. A more detailed review of the history of anthropological studies of alcohol is available (Heath, 1976), showing exponential growth of the literature since 1900.

Drinking and drunkenness have been characteristic of some human beings throughout recorded history. Early sources offer some descriptive clues to such behavior and to attitudes toward it, but few interpretations. Some of the most vivid accounts have to do with the customs of alien populations, often with ethnocentric observations to the effect that such comportment is evidence of their less-than-human quality. Functional interpretations occur early—although they are not couched in such phraseology—as when European colonists attributed drunkenness among Native Americans to their supposed desire to forget their misery, or to escape their barbarous condition. Missionaries, explorers, administrators, and other observers tended to provide vivid descriptions of dramatic and exotic drinking patterns, but they probably neglected to mention many other less spectacular alcohol uses around the world. A few armchair ethnologists compiled encyclopedic gazeteers on drinking customs (as also on various other aspects of culture) during the 19th century, but drinking and drunkenness tended to be seen more as “customs” (with a sort of implicit *deus ex machina* quality of their own) among other populations, and as matters of personal choice in Western culture (e.g., Dorchester, 1884; Morewood, 1838).

Only in the 20th century did anthropology become an academic discipline and did sociology gradually evolve from social philosophy. Theological and moralistic interpretations of behavior began to be supplanted by a variety of psychological views (including psychoanalytic, conditioning, and personality) and by some sociological views (such as reference group theory, enculturation, and roles) long before an integrative systemic view emerged.

Cheinisse (1908) already was talking about anomie as a reason for alcohol-related problems among Jews; by the 1950s a similar interpretation was increasingly being applied to a great many minority (especially colonized) populations (§2.1.1d, 2.3.1). Feldman's (1923) review of theories about

alcoholism showed no appreciation of the relevance of sociocultural variables, although he did oppose the growing demand of eugenicists that alcoholics be banned from having children. An early study of the home brew industry in urban South Africa (Hellman, 1934) provided a number of insightful functional interpretations (§2.9) having to do with leisure, sex roles, ethnicity, and so forth; other attempts to identify what drinking does for people dominated the flurry of topically focused ethnographic reports that appeared in the 1950s and '60s, and functional interpretations probably remain among the most influential models even today when few authors elect to characterize their work as such.

The repeal of Prohibition went far toward lifting the haze of secrecy and moralizing that had obscured many aspects of alcohol use in North America, and the establishment of the Yale Center of Alcohol Studies in the 1940s made research on the subject more respectable. Although E.M. Jellinek, an early and influential member of that group, was a botanist by training, his interest in classical and non-Western cultures and his imaginative curiosity about what is universal and what is unique in human behavior prompted him to speculate about the symbolism of alcohol as a reason for its special place among beverages (Jellinek, 1977), even while he was applying some culture-specific models (§2.1.2) to distinguish, for example, why viticultural Chile had many problems and Italy, where wine is also the commonest beverage, had few. Even in connection with "the Jellinek formula," an epidemiological device based on statistical manipulation of medical data, he was sensitive enough to sociocultural factors (§2.1) to stress that they not only affect but sometimes negate the validity of it, a caveat often ignored by some of his less sophisticated followers.

At the Yale Center, sociologist Selden Bacon very early (1943) stressed the importance of focusing not merely on alcoholism but rather on "drinking behavior." In that paper, he suggested guidelines that are still relevant for delineating what social scientists can and should do in the field of alcohol studies. His own contributions were as much synthetic as analytic; his notes on the important features that distinguish drinking in "complex society" (S.D. Bacon, 1945) were squarely within the sociocultural model (§2.1), emphasizing functional interpretations (§2.9), whereas his more recent analysis of social aspects of addiction (1973) fits with both social organization (§2.4) and symbolic interactionism (§2.7).

A landmark paper by Bunzel (1940), comparing drinking in Indian communities in Mexico and Guatemala, epitomizes the strengths of the sociocultural model, relating different attitudes and practices to different kinds and rates of alcohol-related problems. Functional interpretations (§2.9) figured importantly in her work, to emphasize that positive social integration is as much an outcome of the Indians' style of drinking as are occasional accidents, *faux pas*, and other disruptions. At the same time, Myerson's (1940) emphasis on ambivalence (§2.1.2) presaged a long-term effort by others to sharpen that and the closely related normative model (§2.1); key contributions include Ullman (1958), Mizruchi and Perrucci (1962), Blacker (1966), and Whitehead and Harvey (1974). Also in the early 1940s, anthropologist George P. Murdock, with a few colleagues at Yale, established

the Cross-Cultural Survey (subsequently renamed Human Relations Area Files) as a compendium of ethnographic data that would facilitate large-scale comparative studies. The pioneering essay that became a model in terms of the new methodology — then called cross-cultural research, but subsequently renamed hologeistic (§3.2, 2.3–2.6) — also became a landmark in the field of alcohol studies, the universalistic formulation that stressed anxiety as the primary reason for alcohol use (Horton, 1943). Although the anxiety model has often been tested, adapted, and otherwise developed in various ways by psychologists (including work under the rubrics of "stress," "tension," etc.), it has had little continuing impact in the social sciences, except in connection with specific perspectives relying on notions of anomie or sociocultural deprivation (§2.1.1d, 2.3.1).

The social sciences flourished after World War II, and many North Americans gained a new awareness of cultural differences. The World Health Organization, in searching for a few health problems that would be of interest to its several members, chose alcoholism as one focus, but encountered widely divergent views about it, even among its Committee of Experts. Scandinavians tended to emphasize the public safety aspects, whereas the French were concerned about liver pathology, Americans about economic costs, and so forth. It is noteworthy that the first "official" WHO definition of alcoholism acknowledges the crucial role of the sociocultural normative context: "any form of drinking which in its extent goes beyond the traditional and customary 'dietary' use, or the ordinary compliance with the social drinking customs of the whole community concerned" (WHO, 1951). This recognition of the crucial significance of "customs of the whole community" is all the more striking when it is recalled that there were no social scientists involved in the drafting of the definition, although Jellinek might have qualified as an avid amateur.

Although the viewpoint had been implicit in a few earlier writings, Bales (1946) should probably be credited with establishing the scientific credibility of the sociocultural model. His contrasting of Jewish and Irish beliefs and behaviors with respect to alcohol systematically related those differences to differences in the nature and prevalence of alcohol-related problems in those populations — and his findings not only remain a vital force in the field of alcohol studies but also appear to have influenced the thinking of many North American laypersons.

In academic terms, the publication of an article that reviews the literature on the subject usually constitutes an unofficial sign that the subject has come to be accepted as important. Anthropological and sociological approaches to alcohol studies were probably first reviewed in detail by Ullman (1958) and by Trice and Pittman (1958); the former proposed that ambivalence (§2.1.2) might be the crucial factor that determined how or why normative and other sociocultural differences sometimes result in problems. Snyder's detailed study of Jewish sobriety (1958a,b) provided quantitative support, based on the sociological survey method, for what several others had suggested (on the basis of interpretations of behavior and thematic analysis) about the relevance of norms, reference group, symbolism, and socialization.

Although Ledermann's formulations that came to be called "the single distribution model" were published in 1956, they became influential only when actively promoted in terms of policy implications for the prevention of alcohol-related problems. Among the most prolific proponents have been some scientists at the Addiction Research Foundation of Ontario (see, e.g., Popham et al., 1976; Schmidt & Popham, 1978), although not all of their colleagues are so sanguine (see, e.g., Smart, 1977). The initial interest that WHO had shown in alcoholism was not sustained for long, but a flurry of recent activity relies heavily on the single distribution model (Bruun et al., 1975; Mäkelä et al., 1981; Single et al., 1982); other policy-oriented books tend to emphasize it also (e.g., Beauchamp, 1980; Moore & Gerstein, 1981). Since the 1970s, the single distribution model has gained popularity among some policy-makers who welcome the idea that higher taxes would readily diminish alcohol consumption and thereby reduce alcohol-related problems. During the same period, a few scholars have voiced concern over the degree to which some proponents of that view misrepresent scientific findings in order to make the imposition of controls on access to alcohol appear more attractive (e.g., Heath, in press; Levine, 1984).

The sociocultural model as such has been more often used for interpretation rather than prescription, although reports of the Cooperative Commission on the Study of Alcoholism in the U.S. (Plaut, 1967; Wilkinson, 1970) included a clear set of recommendations that emphasized education and attitudinal change as the most effective way of reducing alcohol-related problems over the long run. Although they are not necessarily characterized as exemplifying the sociocultural model, integrated programs that are designed to influence knowledge and attitudes about alcohol among school children at all levels have been developed in Sweden, Chile, Costa Rica, and elsewhere.

Although relatively few attempts have been made to articulate the sociocultural model clearly and succinctly (Frankel & Whitehead, 1981; Heath, 1980a; Pittman, 1967), some of the related models have been examined in the kind of detail that has had an impact on social science disciplines as well as alcohol studies. For example, a series of studies of drinking in various cultures by Lemert (e.g., 1956, 1962) helped him to shape sociological thinking about deviance. The Tri-Ethnic Research Project at the University of Colorado maintained both conceptual specificity and methodological rigor in comparing neighboring Ute, Anglo-American, and Spanish-American populations with respect to drinking and other forms of deviant behavior; their work (Jessor et al., 1968) is recognized as having contributed significantly to our understanding of social psychology as well as to such perspectives on alcohol as deviance, reference group, labelling, anomie, and socialization.

Anthologies, like review articles, can be viewed as signalling a certain maturity in a field of study. That by McCarthy (1959) emphasized the normative among various sociocultural models. Pittman and Snyder (1962) did not just assemble previously published material but organized original contributions. Everett, Waddell, and Heath (1976) edited the proceedings of a multidisciplinary conference, as did Heath, Waddell, and Topper (1981); the latter volume is unusual in the degree to which it links academic research with

clinical applications. Marshall (1979) made readily accessible much of the ethnographic work published after 1965, and helpful guides to the diverse and scattered literature on sociocultural perspectives about alcohol have been compiled (Heath & Cooper, 1981; Popham & Yawney, 1967). Substantive as well as theoretic issues discussed in this literature are outlined by Heath (1976, 1986a,b, 1987a).

The holodeistic study by Child et al. (1965), emphasizing linkages between child-training and drinking patterns, resulted in the conflict-over-dependency model, although methods and assumptions used in it relate also to the sociocultural, normative, and especially socialization models. It has already been noted that the conflict-over-dependency model was briefly characterized as being incompatible with the power model, although the two have since come to be viewed as essentially complementary (see, e.g., Barry, 1976; Boyatzis, 1976). It is gratifying that the authors of the conflict-over-dependency model have gone beyond their original formulation and continue reworking that rich database in ways that yield fresh insights not only about individuals and societies but also about differences between the sexes (e.g., M. Bacon, 1976). Another major contribution to the normative (§2.1.1) and social learning (§2.8) models is MacAndrew and Edgerton's (1969) review of historical and ethnographic evidence that drunken comportment is shaped more by norms than by pharmacology.

Establishment in 1971 of a National Institute on Alcohol Abuse and Alcoholism within the U.S. Department of Health, Education and Welfare (subsequently renamed Health and Human Services) resulted in a vast expansion of treatment facilities, selective intense efforts at prevention, and some aid to research. The first director, Morris Chafetz, favored education for "responsible drinking," in clear support of the sociocultural, normative, and socialization models; his successor, Ernest Noble, appeared to have been planning a shift toward the single distribution model, but prevention efforts markedly slackened under his successors, John de Luca and Robert Niven. Although NIAAA never explicitly endorsed any particular theory, it was helpful, through the sponsorship of conferences, in fostering codifications of the sociocultural and normative models (Harford et al., 1980) and exchange between sociocultural researchers and treatment constituencies concerned with special populations (Heath et al., 1981).

A first attempt at conducting intensive and extensive research to compare "community responses to alcohol-related problems" in countries that represent strikingly different political and economic systems and "levels of development" was undertaken by the World Health Organization in Mexico, Scotland, the U.S.A., and Zambia, with occasional cooperation from Canada, in the late 1970s (Moser & Rootman, 1981; Rootman & Moser, 1984). A few international and multidisciplinary conferences in the 1980s emphasized cultural variation in drinking patterns (e.g., Babor, 1986; Marshall, 1982). A sociologist's interpretation of why contemporary ethnographers may be guilty of "problem deflation" in their reporting about alcohol use was published simultaneously with several critical responses (Room et al., 1984).

5. BOUNDARIES OF THE THEORY AND MODELS, AND RELATIONS TO OTHER THEORIES

The anthropological theory about alcohol use and its outcomes that seems to be emerging is broad in conceptual scope, just as it is also broad in its applicability to diverse populations. The focus is on social and cultural factors—shared patterns of belief and behavior, especially normative attitudes and values. This emphasis allows one to analyse situations that are remote in time, using a wide range of documentary evidence, as well as to deal with contemporary patterns, whether they are familiar or alien to the observer.

As noted earlier, this formulation of the theory is in large part my own inference from the wide range of models, or representations, that constitute helpful ways in which social scientists have tried to identify key aspects of alcohol-related behavior and to understand some of the ways in which they relate to each other. The editors have observed, and I should highlight for the reader, that whereas the authors of other chapters in this volume are each discussing a more unified theory, I am attempting to organize a collection of models, as a step toward what I have called an “emerging sociocultural theory.” As such, in this section I will be commenting not only on boundaries vis-à-vis other theories, but on relations among models *within* “sociocultural theory.” As a convenience to the reader, this section is organized in parallel with §2, in which the principal elements of the emerging theory were outlined.

It is obvious that this discussion of “emerging anthropological theory” must not only complement but also overlap discussions of “social learning theories,” “conditioning theories,” and other types of approaches to the understanding of alcohol use and its outcomes that are discussed in more detail in other chapters of this book. It may deserve mention that overlap does not imply any academic imperialism but rather is a realistic reflection of the interrelatedness of various approaches to the interpretation of human behavior.

One major difference is that the sociocultural models, to a greater extent than many of the other models, tend most often to be applied to human populations rather than to individuals—to societies, cultures, tribes, nations, classes, ethnic groups, or other populations that may be differentiated as socially significant for any of a wide range of reasons. In an earlier time, such terms as “races,” “minorities,” “nationalities,” “subcultures,” and others would have figured prominently in such a list of categories; in recent times, we see increasing reference to “special populations,” “women,” “youth,” “the aged,” and other categories.

However, a preoccupation with groups does not mean that anthropologists are uninterested in individual variation; they do tend to provide for it in their modelling. How this can be done is discussed below, but it seems important here to mention the general pattern, which differs from most other classes of theory, of both social and individual referents.

Another respect in which many anthropological and sociological models are distinctive is that they allow for a historical dimension. Many are applicable—and some have been applied—to long-term changes in the history of

cultures; to short-term “natural experiments” such as occur with legalized prohibition, strikes in the liquor industry, wartime shortages, and so forth; or even to changes during the lives of some individuals, such as migration, loss of employment, rites of passage, and so forth.

A further characteristic that probably sets most of the sociocultural models apart from many of the models and theories discussed in other chapters is the fact that, with very few exceptions, they are *not* offered to account for alcoholism. Social scientists are not unaware of, or indifferent to, various definitions and the concept of alcoholism; nevertheless, we view it as an extremely small subset within the fascinating field of alcohol use and its outcomes. The subset of phenomena called “alcoholism” (or related subsets such as “drinking problems” or “alcohol-related problems”) can be defined only in relation to other patterns of behavior with respect to alcohol (such as “abstinence” or “social drinking”).

This perspective does not mean that social scientists other than psychologists are uninterested in the problematic outcomes of drinking, but that we tend (sometimes more than colleagues in other fields) to view them in a larger context that also includes non-problematic outcomes.

There is also a recurrent tendency toward eclecticism in the derivation of the concepts that have been used in anthropological and sociological analysis. Among the most obvious cases, social learning is fundamentally based in psychology, as are ambivalence, anxiety, conflict over dependence, and power; discussions of symbolism relate to religion, philosophy, and semiotics as well; models that emphasize norms, deviance, and so forth are linked with social psychology, as is the single distribution model. This kind of transdisciplinary awareness is inherent in the enterprise of making sense out of what human beings do, the fundamental aim of most anthropological work.

5.1 THE SOCIOCULTURAL MODEL

The sociocultural model, as phrased in general terms, can be construed to subsume virtually all of the other models described in this chapter. It also overlaps considerably with some of those described in other chapters. The patterns of belief and behavior about alcohol and its effects that predominate in the sociocultural model are shared by members of a population because of social learning — they are transmitted, rewarded, and enculturated between generations according to the processes explicated in the social learning theory. Insofar as the members of a given society tend to share a common “basic personality” or “modal personality” — not in a literally inclusive sense, but in the sense of predominantly recurring patterns — personality theory is also a relevant complement to the sociocultural model. It can further be argued that political and economic systems are themselves components of more encompassing sociocultural systems; in that sense, political and economic theories may be variants on the sociocultural model, just as they all may be subtypes within a still broader systems theory.

The sociocultural model has relevance in terms of social categories at a wide range of levels. The contrast between “Jews” and “the Irish” has become almost the prototypic example, in which contrasting norms about whether

children should be allowed to drink, the social context in which drinking is initiated, the values and meanings assigned to drinking and drunkenness, and so forth result in strikingly different rates of drinking problems. The nation-state is one kind of population that is often cited, as when the health problems associated with French *alcoolisation* (physical dependence deriving from long-term chronic drinking) are contrasted with the Finnish pattern of trauma from knife fights that accompany rare but violent binges. Religious groups are sometimes compared and contrasted, although the categories may be as narrow as "Unification Church" or "Mormons" on the one hand and as broad as "Catholics," "Jews," or "ascetic Protestants" on the other. It was once popular to write about "Indians," but enormous variation among specific bands, tribes, and other subgroups has dominated in the ethnographic literature in recent years. Much is still written about "blacks" (although class, regional, and other subdivisions would probably reveal significant variation). The literature on "women," "youth," "the aged," and some other "special populations" that have become the focus of burgeoning research during the past decade still offers too little in the way of specification. Illustrative of the confusion that results from such lack of differentiation are the suggestions that recent apparent increases in alcohol-related problems among women in the United States may derive from the stresses (or anomie) that some observers presume are occasioned by their assuming new and unaccustomed roles; or that changing social norms include the expectation that women will drink more and so they have more problems; or that female problem drinkers were there all along but the diminution of social stigma has prompted some to "come out of the closet" or has given physicians the courage to confront them with problems that both had earlier denied; and so forth. Some of the social categories treated in terms of the sociocultural model are enormous and related to systems that permeate the lives of members (e.g., "Muslims"), whereas others are small and relate to only a part of one's life-experience (e.g., "fireman").

Through the process of stereotyping, the sociocultural model is often inappropriately applied — by laypersons — to individuals, as in the deviance and labelling models: for example, "What else can you expect from an Irishman (or Indian, or Albanian)?" Such generalization from patterns that are manifest among populations and their application to individuals is unwarranted and virtually absent from the social science literature.

It is noteworthy that the sociocultural model, when appropriately applied to populations, can often help explain not only differences in the prevalence of alcohol-related problems, but also differences in the nature of such problems. Illustrative of such differences are the predominance of psychopathology among Irish males, cirrhosis among the French, divorce among American women, and so forth.

There are instances in which the limits of the sociocultural model are not at all clear. One example is "the firewater myth" (Leland, 1976) — the proposition that American Indians get drunk faster, stay drunk longer, and are exceptionally aggressive while drunk. Scientists as well as laypersons generally presumed that some sort of "racial" (i.e., genetically inherited physiological) factor was responsible until the middle of this century when the weight of

ethnographic evidence forced recognition of a great many exceptions not only among individuals but among entire Indian populations, some of which are abstinent, temperate, or pacific even while intoxicated. By the 1960s, most scientists had come to view "the firewater myth" as a vestige of racism and to accept the relevance of the sociocultural model in relation to Indian drinking. This is one of the rare instances in history where findings from the social sciences virtually supplanted views that people thought were based on the biological sciences. It was short-lived, however: Since the 1970s, a series of physiological experiments emphasizing the metabolism of ethanol have made it again respectable to talk about possible racial differences in tolerance to alcohol; a good review of the literature is available in Schaefer (1981). Recent indications are that there may in fact be significant differences, previously unrecognized, among populations with respect to the quantity (and even the form) of crucial enzymes (notably, alcohol dehydrogenase) that would account for differential responses to alcohol, including "the Oriental flush," slower dissipation from the body, and so forth; however, since such findings have not been successfully related to drinking, but only to its consequences, the sociocultural model remains useful, especially in terms of "expectancy" or "expectation" as it relates to changes in mood, dexterity, and other variables that appear to be affected by the ingestion of alcohol.

5.1.1 *The Normative Model*

As noted in §2.1.1, the normative model is distinctive primarily insofar as it explicitly cites norms as the content of the process of social learning, the "rules of the game" behind those patterns of belief and behavior that are less precisely referred to in the general sociocultural model. We have already discussed the confusion that occasionally occurs when investigators do not specify which, among various kinds of norms, they are citing. Most of the time, with respect to alcohol studies, the regulative or normative usage predominates, and the systems of reward and punishment that are associated with the social learning theory assure that such normative norms become, for most individuals, the normal norm — that is, most people do what they ought to do most of the time. The "time out" hypothesis is an interesting corollary to this model, emphasizing the fact that many societies seem to afford drunkenness a special status as an unusual interlude in which some of the myriad norms (rules of the game) do not apply.

The normative model would have little relevance from the perspective of biological theories, or from narrowly construed psychoanalytic or conditioning theories, although it could probably be afforded at least some weight by adherents to the other theories discussed in this volume. It is particularly apt in relation to several of the other anthropological and sociological models. Both deviance and labelling relate to those individuals who do not sufficiently comply with the norms of a group; the reference-group model applies to situations in which the norms of one population differ from those of another population that is somehow relevant. In alcohol studies, ambivalence usually refers to the inconsistent quality of socially shared norms themselves; when those norms diverge significantly from those held by an individual, that

individual may suffer from anomie even without being labelled or otherwise classified as deviant.

Although the single distribution model is sometimes juxtaposed as antithetical to the normative model, Ledermann's emphasis on cultural homogeneity and his explication of "the snowball effect" approach the normal kind of norm, acquired through a process of social learning and modelling. Major aspects of the normative model are built into the anxiety model, especially the part (that is usually overlooked) dealing with counter-anxiety, stress based on the expectation of punishment for the violation of norms. The conflict-over-dependency model is another in which norms are of fundamental importance, particularly the discrepancy between the set of regulative norms that governs youthful behavior and others that pertain in adulthood. In the symbolic interactionist model, norms often become inverted, and nonconformity, as defiant behavior, takes on enhanced significance. When norms about drinking and drunkenness are shown to "fit" systematically with norms about other aspects of culture, the author is often offering functional interpretations, which may apply with reference to either the individual or the society as a system.

5.1.1a Deviance. As a subtype of the normative model, deviance has many of the same parameters and stands in a similar relation to many of the other models and theories. There is a strong value connotation, such that deviant individuals and deviant social categories are viewed as not only different but as somehow "bad" in not adhering to the regulative norms of the dominant segment of the population. Alcoholism and drug abuse almost invariably are covered in sociological texts on deviance, together with prostitution, homosexuality, vagrancy, and other widely disapproved patterns of behavior. A few deviant populations are discussed in the alcohol literature, such as bottle-gangs, "homeless men," "the chronic drunkenness offender," or Skid Row, but more often the emphasis is on defiance of prescriptive norms rather than attempting to understand an alternative set of norms. In other respects, the emphasis on deviance closely parallels the normative model in general.

5.1.1b Labelling. When deviance is perceived to be extreme, an offended adherent to the dominant norms may try to dissociate from a deviant. One way is labelling, which accentuates the social distance between the labeller and the person(s) labelled. Such labelling can create reference groups or "outgroups"; the fact that such groups are not normless but rather subscribe to a different set of norms is implicitly recognized in such usages as "the drug subculture," "the brotherhood of the bottle," and so forth. In most respects, what has been said about the normative model in general, and about deviance, also applies to labelling theory. An additional relationship is the interplay with symbolic interactionism, whereby labelling sometimes becomes a self-fulfilling prophecy — if one is repeatedly denigrated as "a drunken bum" or "an alky," there may be little incentive to strive in ways that would dispel the label.

5.1.1c Reference groups. This model refers almost exclusively to populations rather than individuals, even though it derives from the general

normative model. An “ingroup” (or reference group) is a category of individuals who share a set of regulative norms that differ from those of another category or “outgroup,” who, for whatever reasons, stand in some socially significant relationship to the “ingroup.” The persistent idea that “Jews don’t get drunk” is one side of the coin; the obverse is that “drunkenness is a Gentile trait.” Various populations focus on various aspects of culture to distinguish themselves. Even after individuals have habitually adopted many of the ways of a dominant group, they may, on special occasions, still use traditional clothing, language, dances, and other customs as cherished ethnic boundary-markers. Just as drinking temperately may be important to a Jew, a Greek, an Armenian, or a member of some other reference group, abstaining is an article of faith for a Muslim, a Mormon, or a member of some other group. Although the outcomes are different, the psychosocial dynamics are similar for the Quechua Indian, who considers getting drunk an important part of any communitarian religious fiesta, and for the Zambian miner, who feels that he owes himself a drunk with his tribal brothers every payday. A style of drinking and a style of drunken comportment can be cherished as part of one’s heritage, in the same way that a norm of abstinence can be cherished as both an esteemed tradition and a defence against further exploitation by a hostile “outgroup.” Most of what has been said about the normative model in general, and about deviance and labelling theory, also applies to this reference-group model. Ambivalence about drinking tends not to occur in those groups where this model is helpful, although anomie can pose serious problems for any individual who is not very firmly committed to the norms of either group.

5.1.1d Anomie. Anomie is a concept that relates primarily to individuals, although certain cultures may, at given times, be highly conducive to anomie of either or both etiologies (cf. §2.1.1d). An individual whose norms do not fully coincide with those that dominate in the society may suffer the stress of anomie without diverging so much as to seem deviant or to be labelled. Similarly, an individual who has accepted the dominant norms, but who is unable to achieve them, may be anomie. An example of the former might be a Muslim who occasionally wants to earn interest on loans; an example of the latter is an Indian who has moved to the city and wants to become assimilated but whose accent, appearance, lack of references, and/or other features are obstacles to getting a steady job. Anomie is conceived as a kind of severe psychological stress, and drinking to drunkenness is thought to be a frequently used way of reducing the tension, “forgetting one’s troubles,” “drowning one’s sorrows,” and so forth.

In contrast with some of the other variants on the normative model, anomie tends to be invoked more often retrospectively than prospectively. It is not that anomie necessarily drives one to drink, but many drunkards are thought to have become such as a result of anomie. The phenomenon is especially common in situations of transcultural contact, where members of a subordinate group are denied full access to the culture of the dominant group. Once one has rejected parts of one’s traditional culture (e.g., by not fulfilling obligations to kin, by adopting alien dress, or by otherwise violating norms), it may be difficult to regain acceptance. Thus the anomic individual may be

unable to move from one reference group to another and may become stranded "between two worlds." Although most of what has been said about the normative model in general and about deviance and labelling theory also applies to anomie, the predominantly psychological emphasis links anomie with other psychological theories; the concept of anomie itself is close to the acculturative anxiety that figures prominently in the derivation of the anxiety model.

5.1.1e The "time out" hypothesis. The "time out" hypothesis is of little direct importance in relation to alcoholism; its greatest value is in providing a functional explanation of why some populations approve drunkenness. Originally developed by MacAndrew and Edgerton (1969), it is intimately connected with the normative model, inasmuch as "time out" refers to temporary suspension of certain norms that are important at other times. With reference to the development of alcohol-related problems, it is noteworthy that even during "time out" there is always an important "within-limits" clause — some norms may be lifted, but not all. For example, it may be permitted that a person while drunk may exceed the normal limits of sexual propriety — but only to a degree, and certainly not with particular individuals whose kin relationship makes them taboo. The crucial significance of this perspective is that it should temper one's tendency to accept biological (and pharmacological) approaches as crucial in shaping drunken comportment and should underscore the importance of social learning.

Another way in which the "time out" hypothesis provides some clues to the nature of alcohol-related problems short of alcoholism is the nature of drunken comportment. Although it is true that individuals do not always plan exactly what they will do while drunk, there is abundant evidence from many cultures that people often take advantage of the privileged status of drunkenness in order to do things for which they would otherwise be strongly censured.

5.1.1f Ambivalence. We have already noted that the term "ambivalence," as it is used in relation to alcohol use and its outcomes, usually refers to a lack of consistency among the norms that a population holds, rather than to inconsistent attitudes on the part of an individual. Bales (1946) and Ullman (1958) both emphasized that such inconsistency among norms is stressful for individuals who cannot cope with mixed signals; Blacker (1966) linked the ambivalence model with the basic normative model in yet another way, by adding the fact that proscriptive norms can cause more confusion than prescriptive ones; and Whitehead and Harvey (1974) revised that revision of the original formulation by adding a quantitative limit for daily consumption beyond which one would be at risk. The ambivalence model represents one of the few instances in sociology — as well as in alcohol studies — in which a specific hypothesis has been subjected to progressive sharpening by scholars of different academic generations, a history attesting to the importance some people give to ambivalence; sociologist Room's (1976) critique makes the entire sequence an interesting study in the philosophy of science.

Other theories that provide slightly different perspectives on ambivalence include psychological, psychoanalytic, social learning, and political theories. Although this model clearly relates to the etiology of alcohol-related

problems, it sheds no light on what specific kinds of problems might ensue. Neither has there been any effort to discern how it is that so many individuals in societies with such ambivalent norms manage to avoid drinking problems.

5.1.2 *Culture-Specific Models*

As noted earlier, many tentative interpretations have been applied to the specific details of various drinking cultures around the world, by authors who had no familiarity with the wide range of theories about alcohol and who had no pretension of contributing in that sense (cf. §2.1.2). Most of these might equally well be considered functional interpretations, but the ethnographically limited frame of reference set by each author prompted me to list this as a sort of residual category, interesting enough to rummage through if one wishes to learn more about the specifics of drinking worldwide, but of little practical or theoretic relevance to anyone who does not share that curiosity.

5.2 THE SINGLE DISTRIBUTION MODEL

This model is one of the few that very clearly links the biological, psychological, and sociological levels of analysis in terms of its original conception, even though some recent writings have virtually reduced it to a mathematical formula for processing data on sales or taxes of alcoholic beverages. As noted above (§2.2), it is compatible with the sociocultural and normative models in terms of the impact that any individual's drinking habits are presumed to have on those of acquaintances. Neurobiological theory impinges on the model, inasmuch as the upper limits of "the Ledermann curve" (1956) are set in relation to presumed limits of the human body's tolerance for alcohol. Social learning theory (both psychological and sociological) is also linked to it because the "snowball effect" to which Ledermann referred might well be identified with "peer influences," or "modelling" in current parlance.

In various contexts the single distribution model has been juxtaposed with the sociocultural model, as if they were competing to determine which would dominate in the field of alcohol studies, especially with reference to prevention (cf. §7).

5.3 THE ANXIETY MODEL

As it was originally proposed, the anxiety model applied to populations rather than to individuals, and it dealt with drinking and drunkenness but not with alcoholism. Quickly, and with little distortion, it has been generalized to such an extent that it is often cited as the best answer to the fundamental question "Why do people drink?"

Among the other anthropological and sociological models, anxiety fits closely with anomie, conflict over dependency, and the social-defence-mechanism aspect of reference groups (as particular kinds of anxiety); among the psychological theories, psychoanalytic, personality, and social learning are probably of most immediate relevance. Horton's (1943) additional concern about "counter-anxiety" is one of the few clear links with the "time out" hypothesis; he coined the term to apply to the anxiety that a drinker might experience as a result of the social sanctions people would apply to punish

those who violate key norms. Since it is a vector against drunkenness, just as anxiety is a vector favoring it, counter-anxiety links this model with both the normative model and deviance.

There are strong links between the emphasis on sociocultural deprivation and several other theories, as with several of the models discussed in this chapter. The stress on human values is a bridge to humanistic psychology; the relation of those values to self-concept and lifestyle dovetails with personality theory; and the fact that such values — and the depreciation of them — are communicated through social learning makes that theory important in terms of understanding the processes involved. The focus on challenges to a sociocultural system, especially the normative component, underscores the relevance of those models. Inasmuch as members of a depreciated population may be viewed as deviant, be labelled, or adopt defensive strategies of symbolic interaction vis-à-vis members of a dominating reference group, each of those models pertains. The sense of alienation discussed above under the rubric of anomie (§5.1.1d) differs from sociocultural deprivation in that anomie seems not to have been imposed from without. Many of the functional interpretations also relate to this model inasmuch as they emphasize the interrelatedness of values and behavior patterns, and the social and psychological stresses that can result from intercultural confrontation.

Looking at drinking and drunkenness as outcomes of sociocultural deprivation appears to have relevance equally with reference to individuals and to populations. It addresses the question of not only why people drink but also why so many of them drink so much and get drunk so often, with drinking usually viewed as a means of relieving anxiety, and drunkenness a state in which one can, at least temporarily, forget one's troubles.

5.4 THE SOCIAL ORGANIZATION MODEL

Anthropologists have traditionally devoted a major portion of their descriptive and analytic efforts to social organization, as have some sociologists. Nevertheless, Field's (1962) identification of statistically significant associations between aspects of social structure on the one hand and aspects of alcohol use on the other has made little impact. As in any responsible hologeistic study the variables were carefully defined and operationalized. However, the specific variables under consideration are at such a level of abstraction that the motives and actions of individuals seem virtually immaterial. Another shortcoming is that those specific variables have little applicability to complex urban societies. Because of these limitations, it is not clear how the model is related to other models or theories either within or outside anthropological theorizing.

5.5 THE CONFLICT-OVER-DEPENDENCY MODEL

Although the units in any hologeistic comparison are cultures, the conclusions with respect to the conflict-over-dependency model have relevance and can often be applied to individuals. The process of social learning affects every person, and the normative component is emphasized at an early stage in most socialization. The factors that were found to be most significantly

correlated with heavy drinking and frequent drunkenness as patterns typical of populations around the world were discontinuity in the norms of socialization, specifically permissiveness and indulgence of dependency in children followed by the strongest expectations that adults be responsible and independent achievers. Such a conflict presumably produces stress in individuals, with drinking as a convenient way of reducing anxiety and drunkenness as a brief opportunity to take "time out" from the demands of adult life, which seem all the more burdensome because one is unprepared to assume them.

The conflict-over-dependency model is closely related to, but distinct from, the dependency model in psychological theories; the authors (Child et al., 1965) were careful in spelling out their debt to (and differences from) the work of the McCords (1960); there is a logical but much less direct link with the oral dependence factor in psychoanalytic theory or need-dependence in personality theory. In relation to other social science perspectives, it can be construed as a particular case within the general anxiety model.

5.6 THE POWER MODEL

The power model is one of the few interpretations by psychologists of alcohol use that include ample discussion of social and cultural factors. It is also one of the few models that the proponents tested by thematic analysis, hologeistic methods, observation, interviewing, and projective techniques, which illustrate its linkage with personality theory, psychoanalytic theory, social learning theory, and a number of anthropological and sociological models. The important distinction that the authors make between "personalized power" and "socialized power" provides yet further bridges, to political and economic theories and again to social learning. The fact that drinking gives a feeling of power is, at root, attributed to the physiological effect of small amounts of alcohol; so this is at the same time one of the few sociocultural models that includes explicit connection with neurobiological and neuropsychological theories — even though the interpretations of physiology may be misguided.

It is remarkable that, as thorough as the investigators were in devising and applying tests that lend support from the perspectives of various disciplines, some glaring limitations are glossed over. There is brief mention that the model applies only with respect to distilled beverages that have a high alcohol content (McClelland et al., 1972, p.174), even though most of the non-literate populations of the world (including a majority of those whose folktales were subjected to thematic analysis) use only fermented drinks. They also make the point that their model was developed to apply only to males (p.333); a subsequent study stresses that it seems not to fit for females (Wilsnack, 1973).

Like so many of the sociocultural models, the power model incorporates part of the anxiety model, as when "failure to gain social power, or recognition, may turn a man to drinking in order to gain a primitive and narcissistically gratifying sense of personal power" (McClelland et al., 1972, p.197). As such, this model relates more to individuals than to populations, and emphasizes drinking as an act rather than drunkenness or alcoholism. There was some

effort to cast the power model as a corrective to the conflict-over-dependency model, but spokespersons for both groups of researchers now seem satisfied to view them as complementary rather than contradictory (Barry, 1976; Boyatzis, 1976).

5.7 THE SYMBOLIC INTERACTIONIST MODEL

The dramaturgical approach to behavioral analysis, which emphasizes role playing, variations in modes of presenting the self to various audiences, and so forth, is gaining popularity among psychologists, sociologists, and anthropologists, although many do not use the symbolic interactionist label in characterizing their work. It often yields a translation of workaday behavior into the theatrical idiom, which may help observers feel that they have better insight into the motives of the actors. There is usually little attempt at identifying or operationalizing significant variables, and the approach has rarely been used predictively at any level. There are probably some analogues with humanistic and political theories, but this model tends to be applied more for descriptive than analytic purposes. The closest links are probably with the deviance and labelling models, inasmuch as symbolic interactionism, at least in alcohol studies, has usually been applied to patterns of nonconformity (rather than conformity) with the norms that are held by dominant individuals or groups within the given society.

5.8 SOCIALIZATION AND SOCIAL LEARNING AS MODELS

Throughout this discussion of the boundaries and relations of various anthropological and sociological models, many have been cited as having close linkages with social learning. This connection is almost axiomatic, because so much of human behavior consists of rules and patterns that are learned in a social context. In a fundamental sense, among the several definitions of culture a consistent referent is the importance of transmission by learning among individuals within a population. It seems appropriate, however, to make only brief mention of the social learning model here, as it is the subject of an entire chapter elsewhere in this volume (Chapter 7).

5.9 FUNCTIONAL INTERPRETATIONS AS MODELS

There is a sense in which almost any sociocultural theory about alcohol use could be construed as functional inasmuch as it addresses the basic question "How does this behavior relate to other beliefs and behaviors within the culture under discussion?" Such a broad usage would be virtually meaningless, however, and the term is used here to refer to a large number of diverse perspectives, applied to various sociocultural case studies, in which the integrated and integrative quality of shared beliefs and behaviors predominates.

A few examples are in order to illustrate the range of functional interpretations that have been applied to alcohol use and its outcomes. In a South American peasant community where social ties are tenuous, periodic binges with highly ritualized toasting are interpreted as socially integrative, as in the reference-group model. Liquor has been cited as an important tool in the lumpenproletarianization of Arctic populations by exploitative newcomers, an interpretation clearly fitting with economic and political theories but also

clearly functional in terms of those who benefit. The medicinal value of home brew is a function recognized in many societies throughout the world, and so is its nutritional value. A symbolic function associated with alcohol in many societies is the offer of a drink as hospitality; the act of drinking together also often functions as a bond, as when traders seal their agreement on a deal or litigants pledge their acceptance of a settlement with a drink. The ethnographic literature is richly punctuated with such functional interpretations, often proposed by authors who have no familiarity with other such studies or with the vast multidisciplinary literature on theories of alcoholism. Like the culture-specific models, these can be interesting in themselves, and some of them may be generalizable to other situations, but it would be a monumental task to identify them all, much less describe, classify, and analyse them.

6. MAJOR FINDINGS

The emerging anthropological theory of alcohol use and its outcomes combines a number of ways of looking at the relevant phenomena. Such sociocultural models make it easier to identify the many and varied parts of the behavior under study and to understand some of the ways in which those parts relate to each other. The relevant literature is vast, scattered, and diverse (see Heath & Cooper, 1981), but its impact has been substantial. Many of the contributions have been made by researchers who discovered the importance of alcohol only in the course of their research on other subjects, and only a few of them have made it a long-term focus of their professional activities. Furthermore, there is nothing approaching a "standard format" for alcohol research in anthropology or sociology, as there is in many other disciplines.

There is no need to deal with the several classes of models in this brief review of major findings and the subsequent sections on practical implications and research needs; for the rest of this chapter, I will use the term "sociocultural models" as a generic label intended to embrace all of them.

The cardinal significance of sociocultural perspectives has recently been enunciated by a psychiatrist in a way that is more forceful than any anthropologist would have dared to be. Among the major findings of what is probably the largest and longest prospective longitudinal study of alcoholism is the conclusion that "ethnicity (perhaps as a result of attitudes toward alcohol use and abuse)," together with only one other factor ("number of alcoholic relatives"), "accounted for most of the variance in adult alcoholism" in a sample of nearly 400 males who were followed over 33 years (Vaillant & Milofsky, 1982, p.494). "Ethnicity," also called "cultural background" (p.498), was crudely scaled to contrast cultures on the basis of their normative permissiveness concerning drinking by children and drunkenness in men. In sophisticated multifactorial analysis, the cultural factor was measured against several other factors that have been cited as crucial premorbid predictors of alcoholism in other studies, including boyhood competence, childhood environmental strengths and weaknesses, emotional problems, school problems, health problems, social class, sociopathy, and education. Although social scientists are not surprised by

such a finding, it is gratifying to have it highlighted in that authoritative context as another example of the increasing impact that sociocultural perspectives have had in the broad field of alcohol studies.

The empirical findings about alcohol as revealed by anthropologists and sociologists are enormous in quantity, immensely diverse and scattered, and highly uneven in quality. The remarkable diversity of such contributions is reflected in review articles (Heath, 1975, 1986a,b, 1987a) that summarize those sources in terms of categories pertinent to alcohol studies as well as those pertinent to anthropology.

Some of the major findings that have emerged from social science studies of alcohol have become so firmly entrenched that it is easy to forget how recently they emerged. The most fundamental proposition — that social and cultural factors must be considered in combination with biological and psychological factors, if we are to understand patterns and consequences of alcohol use — was not widely accepted by earlier writers, although it is now axiomatic inasmuch as alcoholism is regularly referred to as "a biopsychosocial phenomenon."

A unique contribution from ethnography has been our expanded awareness of the range of variation that occurs in the human experience with relation to alcohol. There is a sense in which such studies comprise a series of "natural experiments" — the same substance, used by members of the same species, with an amazing array of behavioral results (cf., e.g., MacAndrew & Edgerton, 1969; Marshall, 1979). Systematic comparisons can be revealing in terms of the covariance of alcohol-related values, attitudes, and actions with other aspects of the cultural or natural environment (e.g., Bales, 1946; Bunzel, 1940). Similarly, historical studies also constitute "natural experiments" along a time dimension, where changes in drinking patterns and related norms can be related to other kinds of changes (Barrows et al., 1987).

A global overview of the literature on alcohol allows one to recognize a number of important points to which the sociocultural perspective has made major contributions:

- ✓ • Drinking is almost always a social act — in many cultures, drinking alone is unthinkable, and in most, drinking together is an act endowed with strong positive meanings.
- ✓ • Peoples are rarely neutral about drinking — it is often hedged about with a varied lot of norms, to a far greater extent than many other kinds of activity.
- ✓ • Those norms are often endowed with a strong emotional charge, again more than is the case in many other sets of norms.
- ✓ • Such affectively loaded norms include expectations about the results of drinking that are regularly patterned among most members of any given population.
- ✓ • Actual drunken comportment conforms to such patterns, and the fact that it rarely exceeds widely shared limits of propriety indicates that it is strongly affected by social learning.
- ✓ • In most of the cultures where drinking occurs at all, most drinkers have few, if any, alcohol-related problems in physiological, psychological,

social relational, economic, or other terms.

- The phenomenon of dependence or addiction is rare with respect to alcohol, on a worldwide basis.
- Among those individuals who do develop drinking problems, aspects of the cultural context in which they live often play a major role in the etiology of their problem.
- There is no uniform developmental sequence that applies cross-culturally with respect to the way in which various kinds of alcohol-related problems (with the possible exception of certain organic pathologies) manifest themselves.
- For those individuals who seek help in relation to drinking problems, cultural differences can result in potentially harmful misunderstandings; cultural differences can also be helpful in identifying potentially fruitful adaptive strategies for individuals and populations.

More specific findings abound in the anthropological and sociological literature, many of which have been discussed, if only briefly, throughout the rest of this chapter. In view of the fact that most such research does not include detailed specification of hypotheses, strict control of variables, or sampling according to preferred methodological canons, the fact that such major findings have come to be widely accepted is a tribute to the relevance of that research.

7. PRACTICAL IMPLICATIONS OF SOCIOCULTURAL RESEARCH

In view of the diversity of models that have been offered here, it would be difficult to attempt to infer all of their practical implications. Nevertheless, the following brief notes are offered, less in an effort at encyclopedic exhaustion of logical possibilities than in an attempt to highlight a few points that arise out of the sociocultural model and seem to hold greatest promise for relieving or alleviating some of the human suffering that accompanies some kinds of alcohol use.

The increasing attention that has been paid to "special populations" in recent years is a reflection of the widespread acceptance of the importance of sociocultural perspectives:

- The unquestioned epidemiological fact that different populations have different rates of alcohol-related problems and that they also have different kinds of problems demonstrates the reality of difference in human responses to ethanol. It is often the case that those different kinds and rates of problems can be clearly shown to have a direct relationship to specific patterns of belief and behavior.
- Such ethnographic data on cultural differences provide insights that may have immediate utility at many levels of practical concern, such as the search for effective measures for prevention.
- Without a good understanding of the norms of a population, it is difficult to make early identification of individuals who are at high risk for many

kinds of alcohol-related problems, or who are showing early signs that such problems may develop.

- Similarly, a great many of the drinking problems in any population defy diagnosis unless one is familiar with cultural norms and expectations.
- Among the several approaches to treatment that are available, some are appropriate to members of one culture but not another.

A few brief illustrations may clarify these general propositions. On differential rates and kinds of problems, a professional or paraprofessional practitioner in an area with a large Hispanic population should be aware that although men drink heavily, women rarely do, and that boisterous bingeing by groups of men is acceptable but drinking alone is not, nor is any drunkenness in a woman; these norms fit well with norms about sociability and sex roles. With respect to early identification of potential problems in such a setting, a man's lone drinking or a woman's being drunk in any context would be a warning signal. Premature diagnosis can also be problematic, as when the competence is questioned of a clinician who has misinterpreted a man's weekly binges that involve occasional fights and complaints of public intoxication or disturbing the peace; these may be praiseworthy demonstrations of *machismo* that result in elevated status among peers, cause little strain in the family, and in no way presage more frequent or heavier drinking. Even after a Hispanic man has admitted to having alcohol-related problems, the same deeply cherished value of *machismo* may make it difficult for him to accept even the "first step" of Alcoholics Anonymous, to admit to being "powerless over alcohol"—or over anything else, for that matter. Similarly, the kind of confrontations, public confessions, and humiliations that are integral to some group-therapy or halfway-house programs would be repugnant and counterproductive, whereas a vow to a saint or to the Virgin Mary might represent a strong and lasting commitment to abstain.

In recent years, there have been increasing efforts to involve members of various populations in planning and operation in both prevention and treatment fields, so that they might develop "culturally congenial" programs. Some of these have become important within the community, not only successfully dealing with alcohol-related problems but also serving as multi-purpose foci of social activities, preservation and promulgation of the cultural heritage, and so forth (e.g., Cohen et al., 1981). A very different approach is the evolution (or revitalization) of activities that carry a strong component of pride in one's culture and that exact temperance and discipline; although some American Indian dance-cycles have no explicit concern with providing therapy for problem drinkers, some of them very effectively do so (e.g., Jilek, 1981).

It is important to recognize that the various anthropological and sociological models do not apply merely with reference to small enclave populations. At a national level, there is a growing movement in favor of arresting or reversing the postwar trend toward increasing consumption of alcohol throughout Canada. Various policies based on the single distribution model have already been proposed: Per capita consumption is to be lowered by increasing the taxes on alcoholic beverages, restricting the number of sales outlets and the hours they may be open, and raising prices significantly (e.g., Addiction Research

Foundation, 1981; Popham et al., 1978). A similar theme is growing in the United States (e.g., Beauchamp, 1980; West, 1984), although some criticize it by citing the disproportionate impact that such controls would have on the poor and the degree to which Prohibition was flouted in the 1920s. Various educational and religious groups have tended to follow the sociocultural and normative models, stressing education about "how to drink responsibly," "how to decline a drink," and so forth. There have even been some attempts to adapt such campaigns in ways that might have more impact on "special populations."

An awareness of cultural variation in patterns of social relations, in addition to variations in the uses of alcohol, is an important element in planning for prevention and treatment. The distinctive kinds of social networks, in which people participate often include "significant others" whose cooperation is more important than that of members of "the family" as perceived by an unsuspecting social worker. Patterns of help-seeking, folk treatments, and even the definition of what constitutes a serious problem differ markedly from one population to another, and lack of attention to such differences can cause one to overlook or misinterpret problems, to alienate clients, to cause offence by a *faux pas*. Thus, cultural awareness should often be an important consideration in the selection and training of treatment personnel.

No one expects that everyone who is involved in education, prevention, treatment, or other aspects of health and social service with an emphasis on alcohol will become an avid amateur anthropologist. But one can hope that an awareness of the potential importance of sociocultural differences will prompt more people to try to learn at least a little more about the cultures of people with whom they deal regularly, to try to divest themselves of some stereotypes both about other cultures and also about drinking and its outcomes, and to make a serious and sustained effort to find out how things look from the point of view of other persons. A complementary implication is that, where possible, prevention and treatment personnel might be recruited from the populations they are intended to serve.

8. RESEARCH NEEDS

With all that has been learned, much remains to be done toward understanding sociocultural aspects of alcohol use as they relate to both theories and practical applications (cf. Heath, 1980a; Keller, 1979; Room, 1979). It is gratifying that ethnographic methods have gained widespread acceptance, but we still lack research sufficient in detail to let us gain and use knowledge about significant variables even in many of the ethnic groups and other "special populations" within large pluralistic societies, much less in the many small communities around the world that have not yet been studied. For all the studies that we have on heavy drinking and drunkenness, we have remarkably few that explore in depth the values, norms, and patterns of social learning that are involved in widespread abstention, "normal drinking," or "social drinking"; that gap is almost as great with respect to large and complex societies in which most of us live and work as it is in tribal or other groups. It is

not merely a truism to assert that "problem drinking" can better be understood when it is related to unproblematic drinking, or that "excessive," "abnormal," or otherwise deviant drinking and its outcomes imply divergence from some acceptable (normal, or normative) range of variation. As I hope to have indicated throughout this chapter, an advantage of sociocultural studies has been their focus on the full range of drinking behavior.

Although there is now general agreement that social learning plays a major role in terms of drinking behavior and, through expectancy, in the outcomes of drinking, too little attention has been paid to just how it is that people learn what it is they "know" about alcohol (but see Wilson, Chapter 7 in this volume). Only a small portion of the burgeoning research on youth, first drinking experiences, peer influences compared with parental influences, and other themes that depend directly on social learning offers much insight into the processes of communication, example, exhortation, reward and punishment, and so on. Learning about learning would appear to provide the greatest promise for changing what is learned, so that norms, beliefs, and the rest may be altered and future behaviors directed in ways that put individuals at less risk.

In similar manner, we still have much to learn about how people view alcohol and its effects, or what they consider to be alcohol-related problems or alcoholism. It is deceptively easy for those who are deeply involved in alcohol studies to forget that only a few people are aware of the absolute-alcohol parities between a bottle of beer, a glass of wine, and a cocktail; or that many still view liquor as an aphrodisiac. Many behaviors that would otherwise be incomprehensible or appear perverse are understandable and even logical once one is aware of the beliefs and attitudes that others hold. For example, it is not necessarily denial when a person whose norms are different sees no danger in "boozing and brawling," and parents and friends also dismiss it as "just a passing stage, a sign of growing up." To understand such views does not mean that one must accept or endorse them; to ignore them almost inevitably means that one will be ineffectual in dealing with people who do think or act that way.

We need to learn more about the expectancies people have about what alcohol will do, not only to them but for them. Historical and ethnographic studies show clearly that drunken comportment is learned, and related psychological studies have demonstrated that some kinds of behavior are affected more by how much people think they drank than by how much they actually drank. These studies of expectancies are welcome in providing independent support for the general sociocultural and social learning models. More such studies, using sophisticated and rigorous methodology when appropriate (including double-blind and balanced placebo techniques), should contribute significantly to our understanding of why people drink as they do, and why they react to drinking as they do. (Chapter 7, by Wilson, is especially pertinent in this connection.)

With respect to communities, there is still much to be learned about systems of social support and informal channels for help-seeking. However detailed a system may be in place for tracing a client who has entered the formal system of health and other service agencies, there is usually no attention paid to finding out what alternative resources were sought beforehand. The practical

value of such knowledge is clear, both in terms of facilitating and expanding opportunities for early identification of problem drinking and in terms of revealing ways in which agencies concerned with providing health and social services might more effectively become integrated with the local community and reach out to prospective clients.

8.1 ISSUES OF RESEARCH STRATEGY

Some of our needs in terms of research have less to do with new fields of study than with tightening up our methods. There are few anthropologists or sociologists who would not do well to define their terms more precisely and to pay more attention to the operationalization of variables. Comparisons can be misleading if the units compared are not the same order of phenomenon. And, however important intercultural variation may be, greater attention should always be paid to the details of intracultural variation.

It is also important that social and cultural factors not be considered in isolation. Although the ideal of interdisciplinary or multidisciplinary research is often given lip service in alcohol studies, neither happens as regularly as it should. The crucial phenomena of expectancy and social learning, for example, almost require that psychologists and other social scientists collaborate, not only with each other but also with physiologists. The "firewater myth" and "Oriental flush" raised a variety of questions about the interrelations of race, metabolism, diet, expectations, and other factors (cf. §5.1). Genetic studies are handicapped unless one understands how norms about selecting marriage partners affect genealogy, how culturally shaped patterns of food preference directly influence the actions of some enzymes, and so forth.

An important kind of research that has been only rarely applied in alcohol studies is the natural historical approach — that is, dealing with individuals in settings that are normal to them, and in ways that are neither intrusive nor obtrusive. This broad label ranges from microscopic observational studies (reviewed by Heath, 1981; Room, 1981), which have shed light on many aspects of public drinking, to Vaillant's (1983) macroscopic prospective and longitudinal study of health and other variables in the lives of some 400 American men. The difficulties involved in doing research in a way that has minimal impact on the variables under scrutiny are many and complex in the social sciences, but the potential for understanding, in ways that can be helpful in a practical sense, is enormous.

9. CONCLUDING COMMENTS

A broad overview such as this defies summary, and the major topical headings that were suggested by the editors provided ample opportunity to discuss the broad range of subjects that I feel are important with reference to the various sociocultural models of alcohol use. A few points deserve brief reiteration here:

- Although there is no unitary anthropological theory of alcoholism, the several models that have been derived from anthropology and sociology

have had an impact out of all proportion to the fewness of investigators involved.

- An emerging anthropological or sociocultural theory on alcohol use and its outcomes emphasizes the beliefs, attitudes, and values that a population holds with respect to alcohol, and the ways in which they shape behavior that, in turn, affects the individual organism.
- The sociocultural perspective has been firmly grounded in empiricism, with most of the models emerging inductively or being applied post facto to data that were collected without being filtered through whatever selectivity might have accompanied theoretic preconceptions.
- Although many social science research methods do not hew closely to popular canons of scientific rigor, reliability and validity are high, and many findings have been welcomed by colleagues in other fields.
- A rich and varied pool of data that lends itself to alternative uses and interpretations is available, and practical implications of such data are often clear even to a non-specialist.

In attempting to unravel the complexities of alcoholism as a biopsychosocial entity, we cannot afford to slight biological, psychological, or social variables; it is their complex interaction that demands our cooperative attention.

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11. AN ECONOMIC THEORY OF ALCOHOL CONSUMPTION AND ABUSE

Christine Godfrey and Alan Maynard

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1. PRINCIPAL ELEMENTS OF THE THEORY

Consumer behavior has always been of central interest to economists. The analysis of the demand for alcohol, however, raises special issues in addition to those of conventional demand theory. Thus, for example, issues of health, particularly of preventive health, have recently attracted economic analysis (Warner, 1984). The emphasis of economic research has not been on theories to explain individuals' drinking patterns, but the investigation of the social costs of excessive drinking and the economic dimensions of government policies aimed at changing consumption habits. Economic analysis has, therefore, not in general been concerned with devising a theory of alcoholism per se. Economic models have, however, been developed to investigate the factors that influence alcohol consumption and, more recently, how alcohol-related problems are linked to consumption levels.

Economic models of alcohol consumption and abuse can therefore be seen to have two principal elements. The major element in terms of economic content is the modelling of alcohol consumption. The second element is linking models of alcohol consumption with alcohol abuse such as alcoholism. To understand the development of these principal elements it is necessary to consider the methodology available to economists as well as the purposes and uses of their estimated models.

1.1 ECONOMIC THEORY AND METHODOLOGY

Economists are in general unable to test any theories of behavior by

designing controlled experiments to isolate the effects of the separate factors involved (see Orcutt & Orcutt, 1968). Economic models have to be designed to take account of all the simultaneous effects and relationships that occur. The models can only be tested against observable data, which may be scarce and not closely related to the corresponding theoretical constructs. These problems can result in difficulties in sorting out the "cause and effect" between sets of factors and obtaining reliable and precise estimates of the model's parameters. It is not surprising, therefore, that some of those modelling alcohol consumption and abuse resort to fairly casual empiricism. The purpose of this chapter is to review some of the theoretical concepts underlying economists' attempts to investigate theories of alcoholism and to consider the methodology available to economists for testing these theories against available data.

1.2 MODELS OF ALCOHOL CONSUMPTION

Consumers face many choices because consuming one good, say beer, involves foregoing the consumption of some other good. Economic theories of consumer behavior start with the assumption that, given the prices of available goods and services, consumers decide upon the quantities to be purchased by seeking to maximize satisfaction subject to the limit imposed by their income. An individual's demand for any good is therefore likely to be related to its own price, the prices of other goods, the level of income, and possibly other factors. In practice, such models of individual behavior are aggregated to form relationships that can be tested against observed consumption habits of groups of consumers. So, for example, some estimates of the relationship between the consumption of alcohol and its price can be obtained.

Many of the principal elements of the economic theory of consumption are not specific to a particular good such as alcohol. Alcohol does, however, have special characteristics that any model of its consumption needs to incorporate. These distinguishing features relate in part to those special factors, other than price and income, that influence alcohol consumption. Various studies of alcohol consumption have attempted to investigate the influence of advertising, the effect of controls on alcohol retailing, and a number of socio-demographic variables. In many cases the inclusion of such variables has been ad hoc in nature, and the lack of theoretical foundations for models has resulted in the methodological difficulties previously discussed.

A second special aspect is the possibility of addiction or, more generally, habit formation in the consumption of alcohol. The specification and testing of such models has been a feature of alcohol demand studies and is examined below.

An important practical issue is whether it is valid to aggregate these demand functions over all groups of consumers or whether separate functions are necessary for heavy and light drinkers. It could be hypothesized that even if the same theoretical model applied equally to both groups, the values of the parameters of the general model could vary between the groups. For example, heavy drinkers might respond less to price change than lighter drinkers. Few such "disaggregated" studies have been attempted mainly because of a lack of data.

1.3 MODELS LINKING CONSUMPTION AND ALCOHOL ABUSE

It may be felt that without disaggregated models of the type described above, models of average consumption may have little to contribute to studies of alcoholism. Epidemiologists have, however, attempted to provide a link between the average level of consumption of alcohol and rates of alcoholism. Ledermann (1956, 1964) sought to provide a methodology for estimating the proportion of excessive drinkers in a population using the total annual alcohol consumption of the population. He hypothesized that the distribution of alcohol consumption in any population was highly skewed, with the majority drinking relatively small amounts of alcohol, and suggested that this distribution could be represented by a log-normal curve. Additionally Ledermann assumed that there was a fixed amount, equivalent to about 50 pints of beer per day, above which further consumption would soon prove fatal. Only a small proportion of the population would consume more than this amount, and this proportion was set at 1% in all populations and at all times. This restriction allows a unique distribution to be defined and the proportion of excessive drinkers can be determined. If his hypothesis is valid, a knowledge of per capita consumption levels can be taken to be sufficient to estimate the number of alcoholics. The hypothesis has been applied for predictive purposes in support of the view that alcohol control policies that reduce per capita consumption will automatically result in a reduction in the prevalence of excessive drinking.

The applicability and empirical status of this hypothesis has been questioned (see J.C. Duffy, 1977, 1986; J.C. Duffy & Cohen, 1978). It has been claimed, however, that although the detail of the Ledermann theory is not always confirmed by empirical data nevertheless there may be some validity in the notion of a relationship between average consumption and prevalence of heavy use. Skog (1980) refers to this as a "weak" sense of the relationship.

If the Ledermann hypothesis were accepted, a model of alcohol consumption would be sufficient for an economic theory of alcoholism. On the other hand, if the relationship between average consumption and prevalence of excessive drinking only holds in the weak sense, it is important to consider the use and interpretation of models of average consumption.

One argument has been that economists and others should not be concerned only with the very small percentage of drinkers who are chronic alcoholics but with the wider problems of abuse. Moderate drinking or a specific drinking bout may cause problems such as reduced productivity, drunken driving, and public disturbances. These wider abuses and their associated costs may themselves be related to average consumption. The link between the levels of social cost and the level of abuse is unclear. J.C. Duffy (1977) argued that the use of preventive policies designed to change average consumption could only be justified if all consumption could be viewed as a public health hazard. While few have taken an extreme view on the likely harm from alcohol, concern for average levels of consumption has been expressed by a wide variety of bodies: for example, the Department of Health and Social Security (1981) and the Royal College of Psychiatrists (1979).

Another approach has been to bypass all the arguments about the links between alcohol and harm, and, while noting the limitations of the approach, to

use the models of per capita consumption because they are all that is available (Ornstein, 1984).

More recently, however, economists have attempted to tackle this problem by building deterministic models that simultaneously explain alcohol consumption and alcohol harm. The most complete of such studies (Schweitzer et al., 1983) estimated a system of four equations for beer consumption, spirits consumption, alcoholism rates, and alcohol-related mortality. By specifying relationships—for example, with beer consumption being dependent on alcoholism, and with alcoholism being dependent on beer consumption—the interrelationship of these variables could be explored. The system approach, involving a set of jointly dependent or endogenous variables, obviously represents a useful innovative move away from a consideration of alcohol consumption alone, and takes us towards a more complete economic theory of alcoholism.

2. HISTORICAL DEVELOPMENT OF THE THEORY

As indicated, one of the major elements of an economic theory of alcoholism is the modelling of alcohol consumption. In terms of historical development, there have been several strands. The major developments have been related to demand models that are applicable to the study of the consumption of any good, not just alcohol. Many different aspects of these general demand models have been considered. Another strand has been the attempts to identify and estimate the special factors that influence alcohol consumption. The most recent development is to link models of alcohol abuse to these two areas concerned with models of alcohol consumption. These three parts of the historical development of the theory are considered in the rest of this section.

2.1 DEVELOPMENT OF DEMAND THEORY

Demand theory and the analysis of consumer behavior is, and always has been, one of the major areas of economics. The theory of consumer demand is based upon the concept of utility, or the individual's psychic level of satisfaction, which depends on the goods that he or she consumes. The problem for consumers, in economic terms, is to maximize this utility or satisfaction subject to the limitation of their income. At a given set of prices, the goods actually consumed reveal the choices consumers have made. The dependence of demand on prices and consumers' income level is often represented by a demand equation of the following form

$$Q_1 = f(P_1, P_2, \dots, P_n, Y, Z) \quad (1)$$

where Q_1 is the quantity consumed of the first good, P_1 is the price of the good, $P_2 \dots P_n$ are the prices of all other goods, Y is income, and Z is a number of other relevant factors. The set of demand functions for all goods is then a system of n equations with the feature that income is the sum of all expenditures.

Economists, in trying to study demand relationships, have taken one of two courses, estimating demand for each good separately or estimating jointly the demand for all goods. Developments in these two approaches and how they relate to theories of alcohol consumption are now examined.

2.1.1 *The Single-Equation Approach*

The ease of specifying and estimating one equation rather than a whole system has resulted in the main body of empirical demand analysis being based upon the single-equation approach. In general, this approach has been applied rather than theoretical.

As it stands, equation (1) is clearly an inadequate basis for empirical analysis because the mathematical form of the demand function is unspecified. Unfortunately economic theory does not provide much guidance about this aspect of specification. Consequently the functional form of the relationship has been determined more by empirical methods than by theoretical hypotheses about the nature of the expected relationship. Two easily analysed forms are the linear and log-linear relationships, which can be written for, say, the first good as

$$Q_1 = a + b_1 P_1 + b_2 P_2 + \dots + b_n P_n + c Y \quad (2)$$

and

$$\ln Q_1 = a + b_1 \ln P_1 + b_2 \ln P_2 + \dots + b_n \ln P_n + c \ln Y \quad (3)$$

respectively, where $Q_1, P_1 \dots P_n$ and Y are as defined in (1) and \ln denotes the natural logarithm. All other terms of Z in (1) have been ignored for simplicity, but (2) and (3) could be extended to allow such terms to enter in the same way as prices and income. It is obviously difficult (but important) to compare estimates of price and income effects on alcohol consumption that have been obtained from different studies when the empirical models do not have the same functional form and/or differ in the specification of Z . Economists have attempted to tackle this problem by defining indices of the sensitivity of demand that can be computed and compared for various models. These indices are known as elasticities and have become central to the study of demand functions.

An elasticity may be defined as follows: Let one of the variables on the right-hand side of (1) — for example, a price or income — change by a small amount with all the other variables of the demand function being held constant. This small change will cause demand to vary and the elasticity index measuring the responsiveness of demand is defined as the ratio of the proportionate change in demand to the proportionate change in the determining factor. Roughly speaking, an “own price” elasticity for beer of -0.5 would imply that a 1% increase in the price of beer, with all other prices and income being fixed, would lead to a 0.5% drop in the demand for beer. Similarly, if all prices were held constant and income increased by 1%, then an income elasticity of 2 would imply a 2% increase in the demand for beer.

Different functional forms of (1) suggest different interpretations of consumer behavior and are associated with different methods for calculating elasticities from the estimates of the basic parameters of the specified function. The log-linear model (3) is the simplest relationship to discuss in terms of elasticities because they are constant over the whole range of observations and equal to the corresponding parameter in (3). For example, the income elasticity for a log-linear model is the constant c . Other functional forms do not possess constant elasticities. The income elasticity for the linear model (2) is given by $c(Y/Q_1)$ and so varies with the ratio (Y/Q_1) over the sample observations.

The choice of functional form is not the only problem associated with converting equation (1) into an estimatable relationship. There may be a rather imperfect correspondence between the variables that appear in the theory and the quantities for which data are available. Expenditure series are provided for quite broad groups of goods, and some researchers have used aggregates such as total expenditure on alcohol and tobacco (Stone, 1954) and total alcohol consumption (McGuinness, 1980). Alcohol is, however, not one single homogeneous commodity, and attempts to estimate a single alcohol demand function have been criticized. Walsh (1982) re-estimated a disaggregated version of McGuinness's (1980) model differentiating beer, spirits, and wine, and obtained results that were quite different from those reported by McGuinness.

A third problem is related to the number of price coefficients to be estimated. The large number of coefficients and the paucity of data often lead to imprecise estimation. Many demand studies assume the problem can be overcome by aggregating the price effects for all other goods. This solution may seem satisfactory in some studies where there is little interest in distinguishing between the other goods, and their price effects are similar. If separate demand functions for different alcoholic beverages are specified, as suggested by previous work, such aggregation implies that other alcoholic beverages are no more or less substitutes for the type of drink considered than are nonalcoholic goods. This implication seems implausible. It seems important in understanding movements of total alcohol consumption to be able to predict, for example, not only the size of the effect a rise in beer price may have on beer consumption but also what effect it may have on spirits and wine consumption if they become cheaper relative to beer. When such cross-price effects have been included in alcohol studies, the results have not been very satisfactory and the single-equation approach has not been successful in determining the substitutability of various forms of alcoholic beverage (Maynard & Kennan, 1981a).

To conclude, the single-equation approach is an applied rather than theoretical approach and its historical development is difficult to trace. Results from previous studies may not necessarily apply to different populations or time periods. Some lessons have been learned: for example, that it may be inadvisable to consider alcohol as one good. Further development has been limited, however, by the lack of systematic testing of the different formulations of the model; see §4.

2.1.2 Demand Systems

The development of methods for the estimation of complete systems of

demand equations has been associated with more theoretical advances than the single-equation approach. The interest of economists in systems has been motivated in the past by the inadequacies of the single-equation approach to throw light on the cross-substitution effects between goods, as previously discussed. Theoretical developments have often taken the form of considering choices faced by consumers and the nature of their utility (satisfaction) functions. By applying restrictions on the mathematical form of this unobserved utility function, different demand models have been derived. Problems have arisen, however, when attempts have been made to test these theories. It is, of course, important to test the validity of the theoretical restrictions in order to avoid imposing incorrect constraints. The need to estimate an unrestricted model for testing the restrictions, however, limits the level of disaggregation amongst goods. The empirical work has also been satisfactory neither as regards the consistency of the theoretical restrictions on the data nor in identifying an optimum system of demand equations. (See Barten, 1975, and Deaton & Muellbauer, 1980a, for an assessment of previous empirical estimates.)

In general, demand systems have been considered with fairly broad commodity groups, and with only price and income terms included. In these estimates alcohol has been included as one good or even combined with tobacco (Deaton, 1974; Deaton & Muellbauer, 1980b). At the moment, therefore, the systems approach has not yielded insights into the study of alcohol demand.

One development that may be more fruitful in investigating the interrelationship between various types of alcohol is the subsystem approach. This combines features of the single and systems approach. Subsystem models stem from considering the idea of two-stage budgeting. In this approach to budgeting, consumers can be considered to first allocate their budget to broad groups — say food, shelter, transport, etc. — and then only as a second stage to allocate the expenditures on a group to individual commodities within the group. This approach assumes a separate utility framework in which there is a "utility tree" with the broad groups being the main branches representing each of the individual commodities. In the first stage, the factors influencing the allocation of the total budget to broad groups are total expenditure and the group price indices, and the allocation of the thus determined group expenditure is a function of the group expenditure and prices within the group only. The subsystem involves restrictions on the substitution effects possible between goods. In Barten's (1975) words, the approach's "usefulness depends on the ability to classify goods into groups for which the separability assumption may be considered empirically valid" (p.37). With alcohol consumption it is not clear that the "utility tree" branches could be clearly drawn. (See Clements & Johnson, 1983, for an example of this approach applied to alcohol consumption.)

The problem of how to separate goods into the appropriate groups or branches has led theorists to consider other methods of structuring the preferences and substitutability between goods. One approach considered is Lancaster's proposition that the utility function can be stated in terms of the

"characteristics" of the goods rather than the goods themselves. This idea of relating goods by characteristics they possess has attractions, and it has been suggested that the idea may be a useful approach to consider for goods such as alcohol; however, that has yet to be explored (Weedon, 1983).

2.1.3 *Dynamic and Habit-Formation Specifications*

Another important development has been the consideration of specifications that allow for habit in forming demand, and such concepts have been applied to both the single and system demand models. In the simple static single-equation system it is assumed that tastes (and therefore parameters) do not change during the sample period. The inadequacies of such models for some goods, and a growing interest in the importance of taste changes for understanding demand behavior, has led to the specification of habit-formation models in which current levels of consumption depend on past consumption decisions. Much of this work stems from the pioneering analysis of Houthakker and Taylor (1970), and variants of their model have been used for alcohol demand studies.

Houthakker and Taylor (1970) relate demand not only to price and income but also to a variable measuring the existing stocks of that commodity. For items such as consumer durables, it may be expected that the coefficient of the stocks variable would be negative, high stocks from a previous period having a dampening effect on current demand and vice versa. For non-durables, Houthakker and Taylor considered that the stock measure could be viewed as a "psychological" stock variable, measuring habit formation and inertia. In this case, the coefficient of the stock variable should be positive—that is, the more the consumer drank in the past, the more he will want to drink now. Such psychological stock variables are clearly unobservable, and Houthakker and Taylor use mathematical manipulations about the rate of change of this stock to derive a linear relationship in which quantity demanded is related to its own value in the previous period, the corresponding lagged values of price and income, and the current changes in price and income.

Another habit-formation model used frequently is the partial adjustment model (see, e.g., McGuinness & Cowling, 1975). The desired level of consumption is a function of prices, incomes, and other factors as before, but a variety of circumstances prevent consumers from fully adjusting in the period. Actual consumption, as a consequence, differs from the desired level. Combining the adjustment equation with a conventional equation results in an estimatable model including a lagged consumption term (consumption levels one period ago).

M. Duffy (1983) argued that this model may be inappropriate for alcohol consumption because consumers do not face major problems in adjusting alcohol consumption. The form of the equation derived from the partial adjustment approach can, however, be justified on other grounds, and Brown (1952) interpreted the coefficient or the lagged consumption variable as indirectly measuring the habit effect. An alternative set of models has considered the possibility of "addiction" demand. These models have been developed from the work of Scitovsky (1976, 1978) and Marshall (1927), who argued that consumers may respond asymmetrically to changes in market forces; for

example, the change in the quantity demanded may be different in absolute magnitude according to whether prices have risen or fallen. In particular, for a substance like alcohol, consumers may have a tendency to acquire a habit more easily at times of low prices or high income and be reluctant to abandon it when prices rise and income falls. So, for example, considering price changes only, the response to a price rise would be smaller than to a price fall of the same absolute amount. Unfortunately, although Walsh (1982) suggests that such specifications could be applied fruitfully to alcohol consumption data, no such studies have yet been published.

To conclude, Deaton and Muellbauer (1980a) comment on demand analysis in general:

If the final aim of demand analysis is the establishment of a "correct" set of demand functions, complete with well-defined and precisely estimated elasticities, then we are some way from perfection. (p.78)

Their remark seems pertinent to the analysis of the consumption of alcohol. There is clearly scope for exploring the special nature of consumers' behavior in deciding the level of alcohol consumption. These developments may well profit from the work of other disciplines.

2.2 ADDITIONAL VARIABLES

The groups of variables most frequently used in alcohol studies are advertising, licensing and other trade restrictions, and socio-demographic variables. With respect to the development of demand models, there has been a tendency for studies to attempt to include an increasing number of special variables. These do, however, bring additional problems to specifying an adequate demand model.

2.2.1 Advertising

Advertising is undertaken by firms to increase demand for their product. Controversy has surrounded the question as to whether advertising only alters brand shares of a market for a commodity or is capable of increasing the total demand for that commodity. In order to measure the effectiveness of advertising, it is necessary to specify the way in which advertising enters demand models. These measures have varied between studies. Some use the size of current advertising expenditure as a measure (see McGuinness & Cowling, 1975, for further discussion). Others have followed Telser's (1962) work by proposing that advertising should be measured in terms of the number of messages received. A "volume" rather than an expenditure measure of advertising is implied, with the measure taking account of the physical volume of advertising (e.g., column inches) and the audience size.

Another problem is defining what constitutes advertising. Press and TV advertising tend to account for the bulk of firms' advertising. It is less clear whether other activities such as sports sponsorship or other marketing expenditure should be included, particularly in countries where direct advertising is subject to some degree of control.

Consideration of the dynamics of the advertising effect introduces several statistical problems. The hypothesis is that the impact on demand of an advertising campaign is not confined to the current period. To allow for the

effects of past movements, a set of lagged advertising terms could be added to the demand equation. This approach, however, poses a number of problems for estimation. There is no theory to guide the maximum length of lag (i.e., the maximum time period for which past advertising has any effect on sales). Even if the lag length is determined experimentally, there remains the problem that, in general, equations of this type suffer from a high degree of collinearity between successive lagged values, which leads to imprecise estimates.

The final problem to consider is whether there is any simultaneity in the relationship between advertising and consumption. This may occur if, for example, firms adjust advertising budgets in response to changes in sales and other factors. Advertising expenditure would then not be exogenous to the equation, and an estimating technique such as OLS (ordinary least squares) would be biased and inconsistent.

2.2.2 Controls on the Retailing of Alcohol

In most countries the sale of alcohol is subject to some kind of legal control. In general it takes the form of controls on production. Such controls in recent years have not resulted in a rationing of alcohol to consumers but may well have affected the competition between alcohol outlets. If such control leads to inefficient retailing, this result could be regarded as a welfare loss to consumers. The effect of reducing competition may be to raise prices above the level at which they would be if there were free competition among retail outlets. This influence on price levels may affect consumption. The direct effects of retail control on consumption, and therefore the rationale for including variables representing such control in demand equations, are less clear. Two arguments have been proposed for the number of outlets' having a direct effect on demand. A restricted number of outlets may increase transaction costs (e.g., travel to outlets) and thereby reduce demand. Also the outlets (and posters and logos near them) may act as advertising, stimulating demand. The inclusion of a variable measuring the number of outlets does, however, raise the problem of causality, if, as it may be hypothesized, an increase in the demand for alcohol results in an increased number of outlets. Unfortunately, this problem of simultaneity bias has not been investigated in empirical work.

Other controls can be investigated in a different way. The effect of some states in a country lowering minimum drinking ages can be examined directly over time or by cross-sectional studies. Some investigations of such effects are discussed in §3.

2.2.3 Socio-Demographic Variables

Although many sociological and demographic factors have been linked to alcohol consumption, few studies have described in detail how such variables affect consumption. The selection of such variables has in general been based on subjective criteria of plausibility. It is often difficult to interpret the results of the inclusion of such factors as unemployment or tourism in models. Is unemployment, for example, thought to increase or decrease consumption? As unemployment is in general accompanied by a fall in income, an increase in unemployment may result in lower per capita alcohol consumption. This effect would in general be captured by the income term, although the unemployment

rate may act as a proxy for a change in the distribution of income. (See Kennedy et al., 1973, for a further discussion of income distribution and alcohol consumption.) Kitchen (1983) suggests that unemployment is a "stress" variable, and increased levels of stress may result in increased alcohol consumption. These alternative explanations of the effect of an unemployment variable in a demand equation make the interpretation of coefficient estimates difficult.

Other factors such as religion or education may have clearer interpretations, but there are problems with the measurement and number of these variables, which can present difficulties when models are estimated.

2.3 THE SCHWEITZER MODEL

The development of this type of model involves the specification of not just single equations but a complete system. Part of the model consists of demand equations for beer and spirits (Schweitzer et al., 1983). Their specification is similar to that of the single-equation model previously discussed, with a log-linear functional form being adopted. Additional factors included in the equation are tourism, urbanization, temperature, religion, number of outlets, advertising, minimum drinking age, and unemployment. The equations differ from previous models, however, by including alcoholism, which is regarded as a variable to be explained within the system along with alcohol-related mortality and the consumption of beer and spirits. The alcoholism equation required for the completion of the system is specified as being a function of beer and spirits consumption, wine consumption, urbanization, number of outlets, alcoholism rehabilitation expenditure, and unemployment. Alcohol-related mortality was related to the same variables, with alcoholism as an additional endogenous regressor.

Maynard (1983) has previously examined aspects of this model and the considerable difficulties encountered in specifying systems of this type. For example, problems of causation occur in the additional equations of the model relating to alcoholism and alcohol-related mortality. Rehabilitation expenditure is included in both equations, presumably because it is assumed that an increase in such expenditure would reduce alcoholism and alcohol-related mortality, although there is no supporting evidence from micro data for this hypothesis. An increase in rehabilitation expenditure is, however, likely to occur if the number of alcoholics or alcohol-related mortalities is rising. Also the theoretical foundation for including unemployment in the "harm" equations is unclear.

This interesting model should be seen as stimulating further research rather than being the object of too much criticism. If this work is to be developed, there is a clear need to pay attention to other theories on alcoholism and alcohol-related problems. A range of alternative specifications, as with demand studies, is the necessary next step in the development of more complete models of alcoholism.

3. CHARACTERISTIC RESEARCH METHODS

The data available to economic researchers have played a large part in determining research methodology. Data can be available in various forms. Much economic analysis has been based upon observations of alcohol consumption for the whole population over a number of time periods: such observations are referred to as time-series data. An alternative source of information can be obtained from data on individuals or regions at one point in time: such figures are known as cross-sectional data. The models to be used, the factors included, and what hypotheses can be tested are all partly restricted by the data available. A survey of current alcohol consumption for a population, for example, may have no information on individuals' past consumption levels and therefore a habit-formation model cannot be estimated or tested. Cultural factors such as religion may not change sufficiently from year to year to enable the influence of this variable to be measured when a short run of time-series data is all that is available. It is, therefore, worth considering research methods for each type of data set, describing the interpretation and characteristics of the typical demand models. The statistical methods available to test the adequacy of the theoretical models are also discussed.

3.1 TIME-SERIES DATA

Most countries routinely collect consumption and income figures for national accounts. The availability and accuracy of these figures are in part responsible for the relatively large number of models that attempt to describe the changing consumption of alcoholic beverages over a number of years. Even national income data, however, are limited in their coverage. Illegal brewing, smuggling, and legal homemade alcoholic drinks may make figures of total alcohol consumption inaccurate. Also, data on the prices of alcoholic beverages may be limited. Finding a measure that accurately reflects changing prices over time may be difficult. Problems can occur in specifying the factors other than income and price that influence consumption. Some socio-demographic factors do not change sufficiently from year to year to permit their effects to be evaluated with available data. The importance of such omissions depends on the purpose of the study: if the purpose is to predict the effect of policy changes, the omission of factors such as religion that are not usually susceptible to policy pressures may not cause too many problems.

The characteristic method of analysis when the data have been collected is to apply a regression technique to find estimates of the parameters of the equations of the model. Generally the models are linear in the parameters and a simple OLS regression technique can be used. This technique will not always be appropriate: if there is any simultaneity between the regressand and regressor variables (e.g., consumption and advertising), OLS would result in biased estimates. In other instances the theoretical model may involve restrictions that make OLS an inappropriate estimating procedure. For example, some of the specific lag structures on advertising variables make a nonlinear least squares method appropriate (M. Duffy, 1983).

Economic theory leaves many aspects of demand models unclear, and

therefore empirical estimates have to be used to provide guidance on such aspects of the model as functional form. Before any confidence can be given to empirical results, it would seem necessary to follow Hendry's (1980) three golden rules of econometrics and to "test, test, test."

In general, existing studies have not made use of many such statistical tests to judge the validity of their estimate models. The R^2 statistic, which gives a measure of the goodness of fit, and tests of significance of coefficient estimates are used to consider the importance of the variables in determining consumption, but the only test for misspecification generally used is the Durbin-Watson statistic (see Johnston, 1984, pp.314-317). This is a test for a lack of independence in the disturbance term which, if present, would lead to biased estimates of the standard errors, invalid tests, and inefficiency of OLS. A significant value of the Durbin-Watson statistic may, however, be caused by misspecification (e.g., omitted variables), and thus it is often viewed as a general test. If an important variable has been omitted, coefficient estimates will be biased and inconsistent and the usual significance test will be invalid. Some authors of time-series studies have justified the inclusion of a time trend on the grounds that it reflects the influence of omitted variables (McGuinness, 1980). It is difficult to accept this argument because it is unlikely that the omitted variables follow a smooth linear trend. More generally, variables should not be added without some discussion of their role.

3.2 POOLED TIME-SERIES AND CROSS-SECTIONAL DATA

There are difficulties in obtaining reliable estimates of price and income elasticities, and in examining certain other effects on consumption, from time-series data alone, because key variables either tend to move together or exhibit little variation. As well as exhibiting different patterns of variation in regressor values, some cross-sectional data allow the introduction of sociological and other variables thought to influence consumption habits. These variables do not necessarily show a great deal of variation over limited time spans and may not be available as time series. Several studies (e.g., Johnson & Oksanen, 1974) have pooled time-series data with cross-sectional information for various geographical states.

There are, however, some difficulties associated with this approach both with data and appropriate methodologies. The most difficult problem is the so-called border effect, where a possible response to one state's increase in alcohol taxes is simply to purchase alcohol from adjoining states.

The second problem encountered in such studies has been the number and measurement of additional variables introduced. Some studies have included a large number of variables (e.g., Ornstein & Hanssens, 1981), but multicollinearity problems are encountered. The multicollinearity problem arises when series move in line with one another and therefore it is difficult to estimate precisely the effect of any one of the variables. Some of the so-called "cures" for this problem may, however, cause statistical problems of their own; for example, dropping some of the terms of the equation would lead to omitted-variable bias on the resulting coefficients if the dropped variables had an influence on demand.

Some authors have tried to overcome the problems of numbers of socio-demographic variables by principal component analysis. In the Johnson and Oksanen (1977) study, variables were assigned to groups such as education and religion. Principal component analysis was then used to reduce each group of sociological variables and the first two components used in the estimated equation. This procedure is equivalent to imposing restrictions on the coefficients of the model and so may well lead to bias.

The third methodological problem concerns the appropriate statistical technique to use to analyse consumption patterns over time and region. Several alternative techniques have been used, involving different assumptions about the behavior of the error term. For example, the OLS method is based upon the assumption that errors over all observations, times, and regions are independently distributed with zero mean and common variance. Johnson and Oksanen (1977) compared three techniques: OLS; least squares dummy variables with non-random region effects represented by a set of state dummy variables; and a less restrictive generalized least squares approach. In the generalized least squares analysis the error term was assumed to be composed of two effects, a true random error over space and time and a regional error that is invariant over time but varies from one region to another. In the Johnson and Oksanen study, parameter estimates on all but lagged consumption variables were similar whatever the estimation method adopted. These results for Canadian data cannot, however, be generalized to other countries or other time periods.

Difficulties with regression techniques as applied to both time-series and cross-section data led Simon (1966) to introduce a quasi-experimental approach. He studied price movements affected by changes in the state tax rate and calculated the proportional change in state per capita liquor consumption thought to result from the price increases. This calculation assumes that state tax changes are exogenous and not influenced by consumption levels. An important part of Simon's method was to take account of influences other than tax changes on liquor demand by means of control states. This strategy presumes that other factors that influence consumption in one state in any given year will have more or less the same influence on consumption in other similar states in that year. This type of technique was also used by Cook (1981) and Cook and Tauchen (1982) to investigate price changes and changes in cirrhosis mortality and auto accidents. Although the exogeneity of price changes can be tested (see Cook & Tauchen, 1982), the choice and use of control states is more arbitrary and limits the use of the model to considering price effects.

3.3 SAMPLE SURVEYS OF ALCOHOL CONSUMPTION OR EXPENDITURE

A completely different form of data are cross-sectional data based upon sample surveys. Cross-sectional data, in which individual (or household) information is collected, can, like state-level information, be used to supplement time-series analysis. The sets of prices faced by individuals are often quite similar, and so considering demand over families with very different incomes

can assist the estimation of income elasticity. The assumption is that if low-income households had an increase in income, they would attain the same expenditure as the existing high-income households and therefore the elasticities obtained are considered as long run. Cross-sectional studies have other important uses. By considering different price and income responses over different groups of individuals, such as rich and poor, male and female, heavy or light consumers, the differential effects of policy and the consequences of policy can be investigated.

Measures of alcohol consumption obtained from budget and other surveys have been criticized on the grounds of reliability. In many countries, grossing up estimates of expenditure from household budget surveys seriously underestimates the total expenditure given in national accounts. Several reasons have been put forward to explain these discrepancies. One is that such budget surveys do not include tourism, and therefore budget expenditure can be predicted to be below that of national accounts. (See Warner, 1978, for estimates of this effect on American tobacco consumption data.) Such underreporting may not be considered important by a nation's government for considering health effects. A more serious problem arises for analysis if respondents underreport their consumption, particularly if such underreporting varies with the degree of consumption (see Pernanen, 1974); heavy drinkers may, for example, feel ashamed of their consumption levels. Another source of underreporting, however, can be found in the representativeness of sample surveys as regards heavy drinkers. Any projections from such samples to the whole population have therefore to be considered with care.

Different surveys have different measures of consumption that introduce particular methodological difficulties. In budget surveys, for example, many individuals record zero alcohol expenditure in the period of the survey. This renders inappropriate those statistical methods based upon the assumption that the dependent variable is normally distributed. The OLS technique must be replaced by something more complicated, and Tobit models provide a useful starting point (Gomulka, 1984; Keen, 1984).

Other surveys of drinking have used quantity/frequency indices, which take account not only of the average consumption over the period but also how that consumption was distributed. An average expenditure measure may not be able to distinguish, as these indices can, between a person who drank little but often and a person who had infrequent but heavy drinking bouts. Being able to distinguish between different drinking patterns may be a very useful tool in linking consumption with health and public order consequences. Unfortunately such surveys have tended to be occasional in nature and few cohort data, following individuals over different time periods, have been available.

As already stated, the methodology available to the economist is limited. Characteristic research methods used in the study of the economics of alcohol consumption do, however, need to "harden up," as Mayer (1980) suggested was necessary in other areas of economics. There is a need for more data collection, repeated statistical testing of models, and periodic surveys of the state of the "art" to enable some assessment of the contribution of economic theory to studies on alcoholism.

4. RESEARCH RESULTS AND NEEDS

Reviews of empirical work have indicated a wide range in the size and significance of the effects of those variables thought to be important determinants of the level of alcohol consumption (see Maynard & Kennan, 1981b; Ornstein, 1980). Clear comparisons of results are difficult when, as Ornstein (1980) states:

Studies differ in a number of crucial factors which are not held constant, such as differences in time periods and in cultural and social attitudes towards drinking in the countries in question. Moreover econometric techniques lead to differences in results owing to differences in model specification, estimation methodology and quality of the data analyzed. (p.810)

The main purpose of this section is to extend earlier surveys by considering some recent studies for the U.K. The problems mentioned above are obviously reduced by the fact that the results are for one country and moreover the estimates are derived for similar time periods. Attention is also given to some recent work carried out for countries other than the U.K.

The three studies considered are McGuinness (1983), Walsh (1982), and M. Duffy (1983). All presented separate demand equations for beer, spirits, and wine.

McGuinness formulated his model (McGuinness, 1980) in disaggregated terms and used a linear functional form incorporating a time trend. A volume measure of expenditure was related to own price and the other cross-price terms for the remaining two alcohol categories, own and cross advertising effects, the total number of licensed premises, and real income. Walsh (1982) re-estimated the original McGuinness model in disaggregated terms for expenditure and quantity measures of consumption and imposed some restrictions on the price but not the advertising terms. These restrictions imposed symmetry on, for example, the coefficient of the price of beer in the spirits equation and the coefficient of the price of spirits in the beer equation. M. Duffy (1983) conducted a very comprehensive analysis using various functional forms and dynamic as well as static models. The general model related alcohol expenditure of beer, spirits, or wine to own price and all other prices, own advertising and all other advertising, and income. A range of estimates was therefore obtained from this study.

4.1 PRICE

For a comparison of results from different studies, it has been useful to consider elasticities as discussed in §2. Evaluation of elasticities does vary between models. The log-linear model, as previously indicated, produces constant elasticities but for other models the elasticity will vary and access to the raw data is needed to evaluate the elasticity at varying data points. Comparisons of elasticities therefore are restricted to those quoted by the authors.

Table 1 presents these elasticities for the three studies. In all three studies, beer price elasticity was small. For the other two commodities' price elasticities, Duffy's (1983) estimates are consistently higher than the others but still indicate inelastic demand.

TABLE 1

Own Price Elasticities: Values in the table represent the percentage increase or decrease in demand, per 1% increase in price, as estimated by various investigators.

<i>Study</i>	<i>Time period</i>	<i>ELASTICITY</i>		
		<i>Beer</i>	<i>Spirits</i>	<i>Wine</i>
Walsh (1982)	1955 - 1975 (annual)	-0.13* -0.26†	-0.47 -0.45	-0.28 -0.38
McGuinness (1983)	1956 - 1979 (annual)	-0.30	-0.38	-0.17
Duffy (1983)	1963 - 1978 (quarterly)	not available	-0.80 to -1.00	-0.70 to -1.00

* Volume

† Expenditure

Ornstein's survey of U.S. and Canadian estimates of beer elasticity gives results similar to Table 1, with elasticity estimates varying between -0.3 and -0.4. His survey suggested that there is little evidence on wine elasticities but that spirits were price elastic, taking a value between -1.0 and -2.0. It is interesting to compare all these results to the Schweitzer et al. (1983) study, which found both beer and spirits price inelastic. In a simulation of policy initiatives, the calculations made by Schweitzer et al. suggested that a 10% rise in the price of spirits would decrease consumption of spirits by 1.3%.

Until recently the only evidence on cross-price effects came from non-U.K. studies. Johnson and Oksanen's pooled cross-section/time-series study for Canadian data had mixed results, with some of the price effects having the "wrong" (i.e., negative) sign. (A negative sign would suggest that goods are complements rather than substitutes.) The three studies under consideration also had mixed results. Duffy reports unsuccessful attempts to obtain useful results after including more price terms in his model and suggests that the problems may be due to multicollinearity. Walsh imposed symmetry restrictions on the price coefficients, and in both the expenditure and value forms only the cross-price effects between wines and spirits were significant and negative. Possible multicollinearity between variables may suggest that the lack of significance does not justify failure to consider their influences. It is therefore perhaps interesting to note that despite the lack of precision, cross-price effects between wine and beer were also negative but the estimated

cross-price coefficient between spirits and beer was positive. McGuinness's results also in general indicate insignificant cross-price elasticities. Overall, these results do not shed much light on the interrelationships of the demand for different types of alcohol in the U.K.

4.2 INCOME

There has also been considerable variation in the estimates of the income effect. Ornstein's survey indicated that for beer, income elasticity was small or statistically insignificant; no consistent results were found for wine or distilled spirits. The three U.K. studies also indicate divergent results, the estimates of Walsh and McGuinness being consistently below those of Duffy (see Table 2). In all three studies beer had the lowest income elasticities. For spirits and wine the results varied substantially across the studies. Walsh estimated a wine elasticity of 0.5, whereas Duffy found an estimate as high as 2.5. Thus, for example, Duffy's results suggest that a 1% increase in income would lead to a 2.5% increase in the consumption of wine, whereas Walsh's study indicates a change of only 0.5%. If the estimate provided by Duffy is more accurate, then policies directed towards controlling alcohol consumption would have to be designed bearing in mind changing income levels.

TABLE 2

Income Elasticities: Values in the table represent the percentage increase or decrease in demand, per 1% increase in income, as estimated by various investigators.

<i>Study</i>		<i>ELASTICITY</i>		
		<i>Beer</i>	<i>Spirits</i>	<i>Wine</i>
Walsh (1982)	Volume	0.13	1.20	0.51
	Expenditure	0.12	0.99	0.49
McGuinness (1983)		0.13	1.54	1.11
Duffy (1983)		0.80 to 1.10	1.60	2.20 to 2.50

4.3 ADVERTISING

The three U.K. studies all included advertising variables in their model. Duffy found a significant but low advertising effect in the beer equation, the elasticity being 0.07. In the wine equation there was no evidence that

advertising affected demand, and there were mixed results for spirits, the elasticities being small but significant in his two-stage least squares results.

Walsh entered advertising disaggregated by alcohol type. Beer consumption seems to be significantly influenced by beer advertising with again a low elasticity of 0.1; spirits advertising appears to stimulate both wine and spirits consumption. McGuinness's results are also a mixture of significant and insignificant coefficients and, in his own words, "do not suggest that small changes in the levels of real advertising of alcohol will have much of an effect on consumption levels" (1983, p.241).

Similar results of small advertising elasticities have been found in a study of Canadian data (Bourgeois & Barnes, 1979).

4.4 NUMBER OF OUTLETS AND OTHER RETAIL CONTROLS

This variable relates to only two of the U.K. studies, namely those of McGuinness (1983) and Walsh (1982), and in both the number of licences was found to have significant coefficients in the beer equation; the estimated coefficient was also large in McGuinness's results. Moreover, in Walsh's estimates the licences variable was also significant in the spirits and wine equations. The differences between the results reported by Walsh, McGuinness, and Duffy may well be explained by the inclusion of this variable, a possibility that makes further investigation of its influence important.

Canadian and U.S. studies have been able to use differences in regulations between provinces/states to investigate their effects. Ornstein and Hanssens (1981) found the coefficient of on-premise licences per capita to be constantly positive and significant for beer and spirits, but they also point out the problems that might be caused by feedback effects between consumption and licences. They found mixed results for other regulatory variables. Availability on Sunday was found to be important to beer consumption but not to distilled spirits. Also beer consumption, unlike that of distilled spirits, was not significantly different between monopoly and licence states. The minimum legal drinking age was also shown to have strong effects for beer.

Bourgeois and Barnes (1979) found mixed results for Canadian data. Some support was given to the effect of minimum drinking age. Also the number of outlets had some effect on the consumption of beer and wine but not of spirits. Although the difficulties involved in identifying causality were noted by these authors, no statistical tests were applied.

4.5 SOCIO-DEMOGRAPHIC VARIABLES

None of the three U.K. studies included socio-demographic variables. As indicated in other studies, controversy has centred around the role of unemployment and tourism. Unemployment was found to be not only a determinant of consumption in the Schweitzer et al. (1983) model but also an important determinant of alcoholism and alcohol-related mortality. The causation between unemployment and alcohol-related abuse is, however, unclear (see Brenner, 1975, and a comment on this hypothesis by Gravelle et al., 1981).

Other socio-demographic variables such as religion, urbanization, and education have been considered and found to be related to alcohol consumption. These relationships were, however, not of a simple kind. Ornstein and

Hanssens (1981) found that the estimated coefficients of the variables designed to capture the effects of religion indicate that religious affiliation, while inconsistent with heavy drinking of spirits, was positively correlated with beer consumption. Johnson and Oksanen (1974) found mixed results on the influence of education on the consumption of various types of alcoholic beverages, and the only significant effect discovered was that extra years at school tended to be associated with lower levels of the consumption of spirits. Drinking intensity has been found in several studies to be higher in urban areas (Ornstein & Hanssens, 1981 and Schweitzer et al., 1983). The precise nature and magnitude of socio-demographic effects on alcohol consumption are, therefore, quite complex and may be difficult to evaluate.

4.6 EMPIRICAL SUPPORT FOR THEORETICAL MODELS

The results indicate considerable divergence between sets of parameter estimates. Results on aspects of the theoretical basis of models have also varied between studies. Kennedy et al. (1973) found some support for the Houthakker-Taylor (1970) and other habit-formation models, whereas in his study M. Duffy (1983) found the estimates of a Houthakker-Taylor model implausible. Nor is there any clear evidence on the choice of functional form. Duffy did investigate different functional forms by considering the plausibility of results and autocorrelation statistics. Unfortunately, tests designed to detect incorrect functional forms have not been used.

4.7 RESEARCH NEEDS

In the absence of comprehensive statistical testing, the evaluation of results from previous studies is extremely difficult. It is therefore important that, in future, the assumptions of empirical studies be tested thoroughly. The outcome of such testing may be to indicate a need for new models; it is worth noting that, for example, the addiction models discussed in §2 have yet to be applied to alcohol consumption.

The research discussed in this section has been concerned with aggregate alcohol consumption. An important avenue for future research would be to carry out disaggregated analysis in order to explore demand relationships for different age and socioeconomic groups. For example, it would be interesting to investigate whether the young are more responsive to advertising than are other groups of the population. Another important area is the analysis of variance in the price and income elasticities by income group, since these provide information about the differential impact of government policy such as tax changes. A prerequisite for the study of disaggregated alcohol consumption relationships is the availability of a comprehensive and reliable set of data.

5. BOUNDARIES OF THE THEORY

The boundaries of economic models of alcohol consumption and abuse can be divided into two parts. Boundaries exist as regards the applicability of the economic models in explaining alcoholism; other theoretical boundaries

concern the contribution of the demand models discussed here to a more general theory of the economic issues involving alcohol abuse.

The models described here are concerned with predicting movements of population rates of alcohol consumption or alcohol abuse, not with the development of alcohol problems for an individual. It would, of course, be interesting to develop such models including economic factors, if data of a cohort nature were available. Such data may also be more useful in exploring the "addictive" nature of alcohol. One of the boundaries of the economic models discussed here derives from the implicit assumption of rationality and full information as a basis for behavior. For those suffering from chronic alcoholism, many would suggest that they are not free to choose how much they drink. Therefore, although heavy drinkers may be influenced by price and other economic factors, a small subset of chronic alcoholics may be outside such influences. More generally, although habit-formation models have not always been supported by the data, this weakness may reflect the inadequacies of present theories rather than a lack of dependency in alcohol consumption. It is clearly a limitation of current economic theories if, owing to the assumption that tastes remain unchanged, many of the historical cyclical movements in alcohol consumption remain largely unexplained or such models fail to predict the changing preferences for different alcohol beverages taking place at the moment in some industrial countries, such as the U.K.

Models of alcohol consumption are only one part of an economist's concern with alcohol abuse. These demand studies may give some indication of the effectiveness of various prevention policies but the evaluation of the efficiency and social desirability of such policies requires an analysis of their costs and benefits. The precise nature of a cost or benefit in the context of alcohol abuse and prevention policies has been the subject of some debate. Most economists would agree that the costs that fall on others from alcohol abuse should be included in such analysis. These costs of side effects, or "externalities," can be large when, for example, nondrinking persons are involved in alcohol-related motor accidents, alcohol-related fires, or other accidents. Similarly, public order offences can be costly to many innocent parties. Other externalities occur as a result of the institutional framework of the country; so, for example, the cost of medical treatment may be borne not by the alcohol user but through general taxation. More difficult problems arise in deciding how to treat private costs and benefits that accrue to the drinking individual. Mishan (1982) argued that if consumers were considered to have full information and to act rationally, they would act to balance their private costs and benefits, and such calculations had no part to play in a cost/benefit model. Others—for example, Luce and Schweitzer (1978)—have used a model of costs (both private and social) only, implicitly assuming that heavy alcohol use causes costs to the consumers such as illness and premature death without providing any benefits to them.

The role of economists in contributing to the debate on the evaluation of alcohol-related problems is limited, and, as Leu (1983) suggested, a multidisciplinary approach may be required. He concluded that the economist can contribute to theories of alcoholism and alcohol-related problems in two ways:

(1) by providing a normative framework for dealing with alcohol use and misuse and for establishing the appropriate role of government in this area; (2) by providing specific tools, particularly econometric methods and cost-benefit analysis, for evaluating effectiveness and efficiency of government programmes and policies aimed at reducing alcohol related damage. (pp.31-32)

To expand the boundaries of any economic theories on alcoholism requires development of the means to deal with the very specific characteristics of alcohol and how these influence consumers' behavior and consumption decisions.

6. RELATIONSHIP TO OTHER THEORIES

The previous sections have already indicated some of the limitations of using economic models alone. It is therefore useful to consider how such economic models can be related to other theories of alcoholism.

The links between economic and epidemiological models are the basis for many of the applications of the economic models of average alcohol consumption discussed in this chapter. In order to develop more complete models of alcoholism, following the work of Schweitzer et al., more than a specification of economic variables is required, and it is unlikely that such models can be developed by economists alone. Many other economic issues related to alcohol abuse, including cost/benefit analysis and the economic evaluation of treatment, require epidemiological information, and therefore links between economic and epidemiological theories and studies need to be forged.

In order to improve models of alcohol consumption it would be useful to explore the relationship between economic and, for example, sociological models. Johnson and Oksanen (1974) stated, "Typically non-economists have tended to ignore economic variables, while economists have tended to ignore non-economic factors" (p.293). To build models that can more accurately reflect changes in consumption involves exploring the links with other models of alcohol behavior.

Many other models of alcoholism involve investigating individual behavior. The lack of data for individuals, including economic factors such as income, may be one reason that such improved economic models have not been developed. In other economic areas theory and estimation techniques have been developed to explain, for example, individuals' labor market behavior. Linking such economic techniques and other theories of behavior could well be an area of interesting future work, and such studies could help in the development of better aggregate models of consumption and abuse.

7. PRACTICAL APPLICATIONS OF THE THEORY

We have discussed already how well-formulated models may be a guide to judging the effectiveness of alcohol control policies. Attempts have been made

to measure directly the effects of regulation and advertising expenditure. Price elasticities can be considered to give an indication of the effect of a tax change.

Even if further empirical work gives more consistent results, policy predictions obtained from models must be considered with care. Major policy changes, such as a large tax increase, may themselves change structural parameters of the model, and therefore predictions from a model based upon data not including such variability could be misleading.

Nor can models of this sort predict other consequences of policy changes. The predictions of these economic models are based upon "ceteris paribus" (i.e., other things held constant) assumptions. Major changes in policies may draw forth reactions from industry that need further consideration.

Well-constructed and well-tested demand models represent, however, one method of increasing knowledge of alcohol consumption. Although conclusions from these models are limited by problems of interpretation and model comparison discussed in this chapter, provided these difficulties are recognized, the models can at least be one of the tools in the policy-making process.

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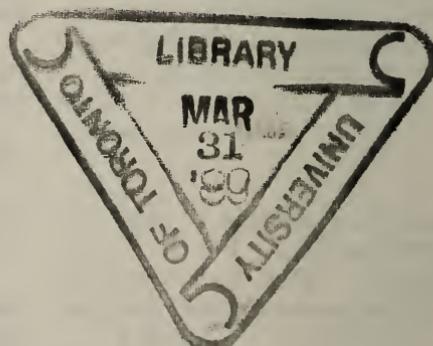
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